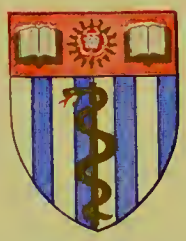
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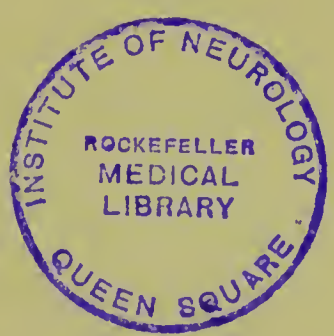
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EPILEPSY

A STUDY OF THE IDIOPATHIC DISEASE





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EPILEPSY

A STUDY OF THE IDIOPATHIC DISEASE

BY

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TO

DAVID FERRIER, M.D., LL.D., F.R.S.,

IN GRATEFUL ACKNOWLEDGMENT OF MANY ACTS
OF KINDNESS.

PREFACE

THE aim of this work is to present a clinical picture of Idiopathic Epilepsy, as a disease which follows a more or less definite course, exhibits distinctive phenomena, and terminates in certain well-known ways. I have avoided all reference to the large and important subject of the mechanism of convulsions, as this would have entailed a discussion of many physiological problems not yet definitely established.

A great and increasing interest in the Epileptic has arisen within the past ten or twelve years, an interest which is largely due to the more recent methods of treating Epileptics, as a class, in Special Institutions. The segregation of persons suffering from this disease in "colonies" has therefore permitted of a more complete study of the natural history of the malady than was previously possible.

My observations upon epilepsy, the results of which are embodied in the following chapters, were based upon the study of one thousand cases, observed partly in my capacity as Visiting Physician at the Colony for Epileptics, Chalfont-St.-Peter, to the Committee and Staff of which Institution I wish to express my thanks and acknowledgments; and partly, in the Out-Patient Department of the National Hospital for the Paralysed and Epileptic, Queen Square. To my colleagues in that Hospital my thanks are due for permission to make use of the notes of some of their cases, more especially in the preparation of the chapter on Prognosis.

Various portions of the work have already appeared, in somewhat modified form, in Volumes 86, 87, and 88 of the *Transactions of the Royal Medical and Chirurgical Society of London.*

I cannot too warmly express my indebtedness to Dr. John Turner of the Essex County Asylum, for the time and labour

which he has expended upon his investigations into the Pathological Anatomy of the disease, and for the chapter on that subject which he has contributed.

It gives me special pleasure to acknowledge the great interest which Dr. Hughlings Jackson has taken in the preparation of the work; and its progress through the press has been largely facilitated by the assistance cordially given me by Dr. T. Walcot of Camberley and Dr. T. Grainger Stewart. To Dr. J. A. Gibson I am indebted for the preparation of the index.

WM. ALDREN TURNER.

Harley Street,
London, *January*, 1907.

CONTENTS

CHAPTER I.

PAGE

INTRODUCTION, - - - - - - - - - - - 1

Definition—General considerations—Inheritance—The convulsive factor—The psychical factor—Conditions underlying convulsions—Conditions underlying dementia—Epilepsy an organic disease.

CHAPTER II.

ETIOLOGY OF EPILEPSY, - - - - - - - - - 12

General prevalence of the disease—Relative frequency in the sexes—Age at onset—The epochal epilepsies—Mean age at onset—Influence of heredity—Collateral heredity—Descendants of epileptics—Signs or stigmata of degeneration—Anatomical, pathological, psychical—Clinical relations of stigmata—General summary.

CHAPTER III.

ETIOLOGY OF EPILEPSY (*continued*), - - - - - - - 41

Determining causes of epilepsy—Reproductive functions—Sleep—Infantile convulsions—Infective disorders—Psychical causes—Trauma—Syphilis—Infantile hemiplegia—Organic epilepsy—Miscellaneous causes—Summary.

CHAPTER IV.

THE CLINICAL STUDY OF EPILEPTIC FITS, - - - - - 65

Classification of epileptic fits—Degrees of convulsion—Prodromata—Fits with peripheral warning; with visceral warning; with head sensations; with psychical warning; with warnings of special sensation; with sudden loss of consciousness—The complete epileptic fit—Immediate sequelæ.

CHAPTER V.

PAGE

THE CLINICAL STUDY OF EPILEPTIC FITS (*continued*), - - 93

Clinical types of epilepsy—Major type—Minor type—Combined type—Serial epilepsy—Status epilepticus—Nocturnal and diurnal forms—Senile epilepsy—Onset and course—Long remissions—Termination.

CHAPTER VI.

THE MENTAL STATES FOUND IN EPILEPSY, - - - - - 118

Epileptic temperament—Pre- and post-paroxysmal psychoses—Acute epileptic dementia—Acute epileptic mania—Transitory delusional states—Automatism—Psychical epileptic equivalents—Psychical epilepsy—Epileptic ambulatory automatism—Epileptic mania—Impulsions—Dreamy mental states—Stupor—Delusional states—Miscellaneous equivalent states—Substitution phenomena—Epileptic dementia—Dementia an integral part of the disease.

CHAPTER VII.

MISCELLANEOUS PHENOMENA OF EPILEPSY, - - - - - 155

1. Neuropathic associations—Paroxysmal headache—Chorea—the tics—Myoclonus epilepsy—Chronic nervous diseases.
2. Accidents due to epileptic fits—tongue biting—scalp wounds—fractures and dislocations—ecchymoses—burns and scalds.
3. Physical condition of epileptics—facies epileptica—general functions—body weight—tremor—cardiac and pulmonary diseases.

CHAPTER VIII.

THE PATHOLOGY OF EPILEPSY, - - - - - - - 165

Pathological anatomy—Changes pointing to defective development—Acute change with swollen nucleus—Chronic change with shrinking and dark staining of cell and nucleus—Changes resulting from epileptic seizures—Neurogliosis—Incidence of gliosis—Sclerosis of cornu Ammonis—Cortical haemorrhages—Changes in thalamus, blood-vessels (angeiomata), cerebellum, medulla oblongata, spinal cord (tract degenerations)—Albuminous exudate in peri-vascular spaces—Changes which appear to act as causal factors of attacks—Thrombi, various forms of—Calcareous or vitreous deposits in the cortex, etc.—Summary of pathological appearances.

CHAPTER IX.

PAGE

THE PATHOLOGY OF EPILEPSY (*continued*), - - - - - 188

Urine—Blood—Cerebro-spinal fluid—Sweat—Thymus gland—Auto-intoxication.

CHAPTER X.

THE DIAGNOSIS OF EPILEPSY AND OF EPILEPTIC FITS, - - 202

Diagnostic features — Epileptic and hysterical fits — Epileptiform convulsions—Aural vertigo—Fits in adult life.

CHAPTER XI.

THE PROGNOSIS AND CURABILITY OF EPILEPSY, - - - 211

Statistics of cure—Prognosis based upon treatment—Conditions influencing prognosis—Heredity—Age at onset—Duration of the disease—Frequency of seizures—Character of seizures—Sex—Cure of epilepsy—General conclusions.

CHAPTER XII.

THE TREATMENT OF EPILEPSY, - - - - - - - 224

Prophylaxis—Treatment at onset of the disease—The bromides; their administration—Other remedies—Miscellaneous methods—Satisfactory cases—Organotherapy—Serotherapy—Treatment during the fit—Status epilepticus—Of the post-paroxysmal phenomena—Hygienic treatment—Education—Dietetic treatment—Institutional treatment—Surgical treatment—Epilepsy and life assurance.

APPENDICES, - - - - - - - - - - 261

INDEX, - - - - - - - - - - - 267

LIST OF ILLUSTRATIONS

CHART I., - - - - - - - - - *facing page* 94

CHART II., - - - - - - - - - „ 96

CHART III., - - - - - - - - - „ 98

CHART IV., - - - - - - - - - „ 100

CHART V., - - - - - - - - - „ 102

CHART VI., - - - - - - - - - „ 104

CHART VII., - - - - - - - - - „ 106

CHART VIII., - - - - - - - - - „ 108

CHART IX., - - - - - - - - - „ 110

CHART X., - - - - - - - - - „ 112

CHART XI., - - - - - - - - - - - *page* 107

FIG. 1.—Betz cell from an epileptic imbecile, showing the characters described in the text which indicate defective development, - - *facing page* 169

FIG. 2.—A similar form of cell from another case of epilepsy with enlarged nucleolus, - - „ 169

FIG. 3.—Betz cell from a case of epileptic idiocy, with enormously swollen 'bladder-like' nucleus, „ 180

FIG. 4.—Capillaries blocked by hyaline clot, - - „ 180

FIG. 5.—Lobulated thrombus lodged in a vessel of the Cornu Ammonis, - - - - - „ 182

FIG. 6.— A cerebellar cortical vein in which is impacted a lobulated thrombus, - - - - „ 182

CHAPTER I.

INTRODUCTION.

Definition—General considerations—Inheritance—The psychical factor—The convulsive factor—Conditions underlying convulsions—Conditions underlying dementia—Epilepsy an organic disease.

EPILEPSY is a chronic progressive disease of the brain, characterised by the periodic occurrence of seizures, in which loss of consciousness is an essential feature ; commonly associated with convulsion, and frequently accompanied by psychical phenomena of a well-defined type ; occurring generally in persons with a hereditary neuropathic history, which shows itself in signs, or stigmata, of degeneration ; running its course uninterruptedly, or with remissions, over a number of years ; and terminating either in a cure, in the establishment of the confirmed disease, in delusional insanity, or in dementia.

It is a malady which has been universally studied since the age of Hippocrates, and has attracted to itself a perennial interest owing to the frequency of its occurrence, the startling picture of its clinical manifestations, and the resistance which it offers to treatment. It is a disease, moreover, the essence of whose nature remains as little determined now, as when Lucretius [1] penned the following graphic account of the classical "grand mal" seizure :

> Oft, too, some wretch, before our startled sight,
> Struck as with lightning, by some keen disease
> Drops sudden : By the dread attack o'erpowered
> He foams, he groans, he trembles and he faints ;
> Now rigid, now convulsed, his labouring lungs
> Heave quick, and quivers each exhausted limb.
> Spread through the frame, so deep the dire disease

[1] Lucretius, *De Rerum Natura*, iii. folio 88, Paris, 1514. Transl. by Mason Good, quoted from Peterson, *Nat. Assoc. Trans.* 1901, p. 11.

Perturbs his spirit: as the briny main
Foams through each wave beneath the tempest's ire,
He groans since every member smarts with pain,
And from his inmost breast, with wontless toil
Confused and harsh, articulation springs.
He raves since soul and spirit are alike
Disturbed throughout, and severed each from each
As urged above distracted by the bane.
But when at length the morbid cause declines,
And the fermenting humours from the heart
Flow back—with staggering foot first treads,
Led gradual on to intellect and strength.

Although our knowledge of the mechanism of convulsions has been enormously extended by elaborate clinico-physiological investigations upon the functions of the cerebral cortex, it has always seemed to me, that the attention bestowed on these studies detracted somewhat from the general consideration of epilepsy as a morbid entity, in which convulsions form only one, though an important, symptom.

It is only during the last decade or two that special study has been given to the chemical pathology of epilepsy, by the analyses of the fluids and secretions of the body, through which interesting and suggestive information has been obtained. During the same time also a revival of clinical interest in the malady has taken place. Our knowledge has been largely augmented by the use of finer methods of staining nerve cells, and their processes, by a more intimate acquaintance with the association tracts and centres of the Central Nervous System, and by the study of statistical and other data obtained from patients treated in special institutions in this and other countries.

In earlier days the convulsion, or fit, was regarded as the sole element of importance in the clinical study of epilepsy; but in more recent years the psychical factor has come to be looked upon as of almost equal importance, and both are regarded as manifestations of a predisposition associated with inheritance.

Inheritance.

The dominant predisposing cause of epilepsy is ancestral epilepsy. My figures, which are detailed on a later page, show that 37·2 per cent. of the cases have a direct inheritance of

this character: and that if other neuropathic conditions, such as insanity and alcoholism, are added, the percentage of those with a hereditary history rises to 51 per cent. It is probable that these figures are considerably below the actual percentage of cases with hereditary predisposition, as a closer analysis shows, that a difference of 19 per cent. exists between the cases which are seen in private, where the family history is better known, and in hospital practice—a difference which is sufficient evidence of the extent to which hereditary influences are either forgotten or unknown. The figures also vary materially according to the authority by whom they are collected, a variation of from 25 to 66 per cent. being found as expressing the proportion of the hereditary element in the causation of epilepsy.

Epilepsy, therefore, is a malady indicative of a family neuropathic degeneration, and is closely allied to such other neuroses as insanity, alcoholism, and hysteria. A neuropathie heredity is revealed in many ways, but sure signs of it are found in what are known as "stigmata" of degeneration. These may be either structural in character, or psychical peculiarities of individual temperament and disposition. There is a close relation between the stigmata and the clinical eourse and features of epilepsy. When the disease begins early in life—under five years of age—structural stigmata are frequent and well-marked, and indicate the intensity of the neuropathie heredity—a point which receives confirmation in the fact that other hereditary degenerative conditions, such as stammering, backwardness in walking and talking, delayed dentition, deaf-mutism, and imbecility, are observed during the same period. The close association also between the presence of stigmata and the degree of mental infirmity leads to the same conclusion.

These observations point to the hereditary influences at work in the causation of epilepsy; but a brief reference may be made to the effect of conditions acquired by the parent, such as the drug habit, alcoholism, syphilis, gout, and tuberculosis.

Many writers hold that "drug habits" are symptoms of neuropathic heredity, and as such have the same relation to epilepsy as other hereditary factors—a position which it is difficult to combat. Congenital syphilis will be shown in the sequel to have little, if indeed any, effect in the causation of epilepsy: gout and tubercle do not seem to stand in any relation

other than through the debilitating influences which their coexistence with epilepsy entails.

The Convulsive Factor.

The only entirely characteristic symptom of epilepsy is the periodic recurrence of attacks of loss, or impairment, of consciousness, sometimes accompanied by convulsion. But even this definition requires modification, as there are occasional inter-paroxysmal phenomena in epilepsy without loss of consciousness, and some impairment of consciousness may be found in epileptoid states, such as aural vertigo, and hystero-epilepsy.

The doctrine originally propounded by Herpin[1] has been adopted in this work as the basis of the description of the convulsive phenomena of epilepsy, viz., that the incomplete attacks, embracing the "aura-sensations," cramps, spasms, and partial convulsions of the inter-paroxysmal periods, are the initial phenomena of the complete seizures, which have been arrested at some stage of their course. The most logical conception, therefore, of the spasmodic and convulsive symptoms of epilepsy lies in regarding them as links of a chain, and not as isolated features in a convulsive disorder. In consequence, there may be found in the same person all gradations of paroxysms from the simple aura, through the incomplete attack, up to the fully developed complete epileptic fit. Applying the same principle to the clinical groupings of fits, there may be observed all degrees of combination of seizure, from short series of incomplete minor or major attacks, through serial outbursts up to the complete picture of the status epilepticus.

As the minor type of fit is merely an aborted, or modified major fit, so the serial outburst is a modified development of the status epilepticus. Short series of minor and major fits are gradational stages towards the more fully developed serial epilepsy, which is in turn a subacute status attack.

Like other disorders, epilepsy, through its convulsive symptoms, may present both an acute and a chronic development. There are rare instances in which recurring attacks of the status epilepticus form the clinical type of the disease; on the other hand, an attack of status epilepticus may be the acute commencement of epilepsy, which is continued in a chronic

[1] Herpin, *Les Accès Incomplets d'Epilepsie*, Paris, 1867.

form by the recurrence of ordinary major or minor seizures; or an attack of status may develop at any period of the confirmed malady, from an accidental cause, such as a fall or blow upon the head, child-birth, an acute infective disorder, or some mental excitement; and, fourthly, status epilepticus may be the ultimate development in many old standing cases of the confirmed disease.

Epilepsy is most commonly found in a chronic form, with periodic seizures persisting over long intervals of time, but sometimes showing prolonged and characteristic remissions. Eventually, however, except in the rare cases in which a permanent arrest takes place, the fits increase in number and severity, and the mental condition tends towards permanent delusional states and dementia.

The Psychical Factor.

The recognition of the psychical factor in epilepsy has materially widened the range of epileptic manifestations, and some authorities have relegated the convulsive element to a subsidiary position. Aschaffenburg,[1] indeed, has stated that neither vertigo, nor convulsion, should be looked upon as a cardinal symptom, but rather "periodic fluctuations of the psychical equilibrium," which may lead to disturbances of consciousness, and may, or may not, be accompanied by convulsion. How far periodic variations of the psychical state, with impairment of consciousness, should be regarded as epileptic will be more fully discussed in a later chapter; but it is now a generally accepted doctrine that certain psychical paroxysmal phenomena occurring in epilepties may replace convulsive seizures, and form what have come to be known as psychical epileptic equivalents. These psychical equivalents I hold to be nothing else than the temporary psychoses which are commonly found to precede, or succeed, ordinary epileptic seizures; for I have never seen a psychical equivalent condition which had not its counterpart in the pre- and post-paroxysmal psychoses of epileptics; and their presence, both as post-convulsive and as equivalent states, is frequently observed in the same individual. Under this heading there will fall to be described those transitory phases of acute mania, of stupor, of automatism, and the various perverted and bizarre

[1] Aschaffenburg, *Arch. für Psychiatrie*, Bd. 20. p. 955.

actions of the interparoxysmal periods which come more particularly into the domain of legal medicine.

In addition to the paroxysmal psychical phenomena, there is observed an almost constant and characteristic inter-paroxysmal mental state, presenting features of a uniform and constant kind. The general features of this condition consist primarily, and most commonly, of a defect of memory, more especially for recent events: but there are also found, as constituent symptoms, all degrees of mental impairment up to pronounced dementia. Observations will be brought forward, and arguments will be advanced to show, that the mental characteristics of the inter-paroxysmal periods should be regarded as integral features of the disease, although modified to some extent by the duration, frequency, and character of the fits.

The Conditions underlying Convulsions are usually looked upon as associated with the cerebral circulation, and rather with an anaemic than a hyperaemic condition. The available evidence upon this point may be briefly mentioned. Ligature of the carotid arteries in animals has been found to give rise to loss of consciousness and convulsions (Astley-Cooper, Kussmaul and Tenner), and obstruction to the cerebral venous circulation also, though less quickly, may lead to spasm (Hermann and Esher, Ferrari).

Leonard Hill[1] has shown that the immediate effects of ligature of the cerebral arteries varied, in different species of animals, and according to the extent and degree of the vascular occlusion, thus: "If the cerebrum be rendered bloodless, loss of consciousness and motor paresis arise; if the mid-brain and medulla are cut off from the circulation, spasms result; vaso-motor spasm producing a high blood pressure, respiratory spasm and a slow heart by spasm of the vagus centre." The effects of sudden and simultaneous compression of both carotids in man produces loss of consciousness. Kussmaul and Tenner compressed the carotids in six men, in two of whom general spasms and the phenomena of an epileptic fit ensued. Schiff and Hill have produced clonic spasms of the opposite side of the body by digital compression of one carotid artery. Hill thus describes the phenomena as observed in himself: "The

[1] *Cerebral Circulation*, 1898, p. 124. This contains an ample bibliography on the subject, from which references have been taken.

first effect on applying the compression was a sensation in the eye in the same side; then there followed a sensory march of formication down the opposite side of the body. This began in the fingers, spread up the arm, then down the leg. Finally, clonic spasms of the head occurred, accompanied by an intense feeling of vertigo and alarm"—a clear description of a Jacksonian fit.

From the above observations, it is seen that sudden anaemia of the brain may give rise to convulsive spasms, but whether they are in essential features analogous to those of genuine epilepsy is a matter of divergent opinion. The true Kussmaul-Tenner fits are bulbar ("lowest-level") fits, and like Brown-Séquard's fits in guinea pigs, and probably also the fits produced by absinthe injections, are epileptoid, rather than epileptic phenomena.[1]

The doctrine that cerebral anaemia may be the exciting cause of convulsions has led to the view that, at the onset of an epileptic discharge, a state of spasmodic vaso-constriction occurs. There is, however, no evidence obtainable of the spasmodic constriction of the cerebral blood-vessels. Hill has stated that every experimental method employed to detect vaso-constriction has failed; and examinations of the fundus oculi, by aid of the ophthalmoscope, at the commencement of epileptic seizures have also failed to show any vascular change of this character occurring in the retinal arteries.[2] It would appear, therefore, as if there was no fundamental proof of the existence of vaso-motor spasm, as a cause of convulsions.

A method of treatment of epilepsy was introduced in the belief that a condition of cerebral anaemia was the cause of the convulsive seizures. The practice consisted in removal of the cervical sympathetic ganglia, with a view to convert an anaemic into a permanent hyperaemic cerebral state, from paralysis of the vaso-constrictor nerves which form a part of these structures. This operation, it need scarcely be said, was not followed by that improvement in the disease, which on theoretical considerations might have been expected.

Influenced to some extent by the effects of sudden anaemia of the brain, as a cause of convulsions, Dr. John Turner has investigated more especially the condition of the smaller blood-vessels in the brains of persons dying from epilepsy, and has

[1] Hughlings Jackson, *Lumleian Lectures*, 1890.

[2] Gowers, *Medical Ophthalmoscopy*, 1890, p. 202.

described his observations in the chapter upon Pathology contained in this volume. The changes, which he has observed and recorded, consist in the formation of coagula in the veins, capillaries, and arterioles of the cerebrum and cerebellum. The clots appear to be formed by the accumulation and amalgamation of blood plates. Suitable staining reagents show that these coagula contain phosphorus, probably as a constituent of their nucleo-proteid. These clots eventually undergo degenerative changes, and in process of time become absorbed. The chief effect of these thromboses, which are formed extensively throughout the brain, is to deprive the cortical grey matter of the arterial blood necessary for its proper function, and to induce a condition of blood stasis. If the coagulation and obstruction occur in the veins, as is commonly the case, the same result is brought about as if they occurred in the arterioles, viz., arrest of the arterial blood supply to the corresponding cortical area. Apparently, as a result of the deprivation of arterial blood, the large pyramidal cells of Betz show characteristic alterations, such as a large, swollen and clear nucleus, similar to what has been described by Mott,[1] from the experiments performed by Hill upon ligature of the carotid arteries in dogs, to which reference has already been made. As a result of these observations, Dr. John Turner is of opinion that convulsions would appear to be due to the deprivation of arterial blood in cortical areas, coinciding with capillary and venous stasis, in persons hereditarily predisposed to nervous instability.

There are certain facts which would favour this view as a not unlikely explanation of some convulsive seizures occurring in epilepsy. First, the marked frequency of epileptic fits at night, especially during the hour or two immediately after falling off to sleep, when the cerebral blood-pressure is at its lowest; and, secondly, the frequency of epileptic fits of a severe character in association with acute infective disorders, such as pneumonia, enteric and scarlet fevers, in which there is an increased coagulability of the blood and a tendency to venous thrombosis. In this category also may be placed the puerperium—a period which is notoriously prone to the incidence of epileptic fits.

The consideration of this subject leads on to the possible production of some fits by a state of *autointoxication*—a cause

[1] Mott, *Croonian Lectures*, 1900.

of epileptic convulsion which has received considerable attention and discussion, but little satisfactory support, during recent years. This subject is dealt with more fully in Chapter IX., but it may be incidentally mentioned that there are observations pointing to a toxic condition of the blood in some of the clinical types of epilepsy. First, the observations of Wooldridge showed that intra-vascular clotting occurred in cases of infection, probably as a result of the liberation of nucleo-proteid, and that the injection of nucleo-proteid experimentally caused clotting in the blood-vessels during life. Intra-vascular nucleo-proteid thrombosis has been found in the smaller blood-vessels of epileptics, and is described in the chapter on Pathology, and from this it has been argued that some forms of epileptic fits may arise indirectly from toxaemic causes arising within the body. Secondly, the observations of Lewis Bruce[1] point to the existence of a hyper-leucocytosis in the blood of epileptics, when examined after a serial outburst, or an attack of the status epilepticus. Hyper-leucocytosis is regarded as a symptom of toxaemia, and is especially well marked in some forms of acute insanity of toxic origin.

Bearing upon the toxic causation of epileptic fits are the observations of Clark and Prout,[2] which confirm the view that epilepsy is essentially a cortical sensory phenomenon with motor manifestations. These observers have stated that "the most striking changes presented by the cerebral cortex in epileptics are found in the cells of the second cortical layer. The cells are swollen in many instances to twice their normal size, the nucleus being especially large and granular, while the ultimate disappearance from the cortex of cells, so seriously altered, is to be inferred." From the constancy of the cell changes, from their general distribution in the second cortical layer, and from their presence in mild as well as severe types of the disease, these authors infer that epileptic convulsions are evidence of a diseased state of the sensory elements of the cerebral cortex.

Various other conceptions upon the auto-toxic production of epileptic seizures, such as Haig's uric acid theory, Krainsky's carbamate of ammonium, Donath's cholin, and Ceni's cyto-toxins, do not require further consideration in this place, as there is no certain proof that such conditions underlie the

[1] Lewis Bruce, *Studies in Clinical Psychiatry*, 1906.

[2] Clark and Prout in Spratling's *Epilepsy*, p. 325.

onset of fits; the evidence, on the other hand, points more readily to these substances being the effects, rather than the cause, of epileptic seizures.

Conditions underlying Epileptic Dementia. The repeated occurrence of the changes, which induce the periodic seizures of epilepsy, lead, in process of time, to alterations of a permanent character in the structure of the cerebral cortex. These appearances will be detailed in Chapter VIII., but may be stated to consist mainly of an increase of the neuroglia, more especially of the outermost cortical layers, often having a patchy distribution, and involving more particularly the region of the cornu Ammonis. The sclerosis of the epileptic brain is due less to a proliferation of the glial tissue than to a disappearance and replacement of the nervous elements, whereby the neuroglial fibrils become closely approximated and more conspicuous. This neuroglial sclerosis has been described by most writers upon the pathology of epilepsy (Féré, Chaslin, Alzheimer), and Binswanger has referred to its existence, especially in cases of old standing epilepsy with well-marked mental deterioration. Atrophy and sclerosis of some part of the central nervous system were found in considerably more than half the cases examined for this research.

It would, therefore, appear from the observations, which have been made upon the structural changes found in the brains of persons dying from epilepsy, that whatever may be the immediate exciting cause of individual fits—primary thrombotic occlusions or cytological changes, directly, or indirectly, induced—sclerosis of the neuroglial tissue eventually develops, replaces to a large extent the cellular cortical elements, and leads to atrophy of the cerebral convolutions. Hence there ensue, in the later phases of the disease, all those mental and physical signs of impaired cerebral function, so well known to those conversant with the symptoms of the confirmed malady,—the pronounced mental deterioration, the slow gait, slouching attitude, and the tremulous, slurring speech, a clinical picture not unlike that observed in the later stages of paretic dementia.

Epilepsy an Organic Disease of the Brain.

Reference has been already incidentally made to the conditions underlying the two most prominent symptoms of epilepsy,

the convulsive and the psychical factors. It has been already stated, and further evidence will be brought forward in greater detail in future pages to prove, that both the fit and the inter-paroxysmal mental state are due to pathological changes, respectively of a transient and permanent character. The morbid conditions, which give rise to the convulsions, eventually lead to those which underlie the permanent dementia: the arterial and venous thromboses (J. Turner), or the cytological changes of the cortical cells (Clark and Prout) are the forerunners of the cortical sclerosis of the later stages of the disease.

Not only are the changes, to which the convulsive and psychical symptoms are directly attributable, evidence of the organic nature of the malady, but the inherited neuropathic predisposition is revealed by well-marked structural signs or stigmata of degeneration. Neuropathic stigmata are seen in abnormal development of the head, face, palate, ears, body and limbs; and in the brain itself anatomical evidence may be found of structures, which it is believed indicate the inherited degenerative disposition (Roncoroni, Lugaro, Kaes).

Allen Starr[1] refers to the assigned causes of epilepsy as arguments in favour of the organic nature of the disease in many instances. He points to the frequency of trauma of the head as a cause of convulsions, to arterio-sclerosis as a common association of the late epilepsies, and to the frequent co-existence and causal importance of mal-development of the brain in the epilepsies of childhood and youth. Moreover, many convulsive disorders, indistinguishable from idiopathic epilepsy, are found in association with organic lesions of the brain, such as porencephaly, cerebral syphilis, and tumour formation.

I submit, also, that the unsatisfactory results of treatment in the majority of the cases of epilepsy, the overwhelming number of epileptics who become victims of the confirmed disease, and the progressive character of both the paroxysmal and inter-paroxysmal symptoms, point to the existence of an organic change underlying its symptomatology, a change which has been described by most workers upon the pathology of the disease.

[1] Allen Starr, *Journ. of Nerv. and Ment. Diseases*, 1904, p. 145.

CHAPTER II.

ETIOLOGY OF EPILEPSY.

General prevalence of the disease—Relative frequency in the sexes—Age at onset—The epochal epilepsies—Mean age at onset—Influence of heredity—Collateral heredity—Descendants of epileptics—Signs or stigmata of degeneration—Anatomical, physiological, psychical—Clinical relations of stigmata—General summary.

SOME GENERAL ETIOLOGICAL CONSIDERATIONS AND THE INFLUENCE OF A NEUROPATHIC PREDISPOSITION IN THE CAUSATION OF EPILEPSY.

1. The General Prevalence of Epilepsy.

It is difficult to arrive at an accurate estimation of the frequency of epilepsy and its prevalence amongst the population of different countries. No census for this purpose has been taken in the United Kingdom, while those made abroad have been only partial. Epilepsy, moreover, is a disease so closely related in the early years of life to idiocy and imbecility, and in the later years to dementia, that it is almost impossible to separate them satisfactorily for statistical purposes. In a number of persons the disease persists throughout life unrecognised, while in others its existence is concealed, and no reference would be made to it in a census taken for the purpose of ascertaining the total number of epilepties. There are also numbers of children suffering from recurring convulsions, without doubt epileptic in character, in whom the true nature of the malady is not suspected.

Statistics taken with the object of ascertaining the total number of epilepties, are accordingly based upon those cases which are known to be epileptics, and are resident either in Homes, Special Institutions and Asylums for the Insane, or are under the care of the Poor Law Authorities—a method of investiga-

tion which clearly does not represent the total number of epileptics in the population.

In countries having a compulsory military service, valuable information upon the prevalence of epilepsy amongst the young male population is forthcoming. By this means a fairly accurate estimate has been formed of the general prevalence of the disease in France, Italy, and Germany; and the figures, which are reproduced here, have been based upon such investigations. From the figures given by Burlureaux,[1] and quoted by Féré, I have extracted the following averages as regards the prevalence of epilepsy amongst the French recruits: For some of the Southern districts of France, 2·9 per 1000; for Paris, 1·5 per 1000, and for the Northern and Western Provinces, ·7 per 1000, giving a total average of 2·5 per 1000 recruits over the greater part of France. In Italy Morselli[2] states that out of 10,000 young men who presented themselves for military duties, 11·5 were rejected owing to epilepsy, which gives a ratio of 1·15 per 1000. In Prussia Binswanger[3] relates that in the year 1895, 2·9 per 1000 recruits were rejected on account of epileptic fits.

The following additional facts and figures upon the prevalence of epilepsy in various countries have been gleaned from divers sources. Peterson[4] estimated the ratio of epileptics to the population at large in the United States of America at 2 per 1000. Kolle[5] put the proportion at 1·3 per 1000 for the whole of Switzerland; and Bircher[6] stated the ratio at 2·5 per 1000, more particularly in the Canton Aargau. In a census taken in 1882 of the population of Mecklenburg Schwerin, Tigges found that one person in every 855 of the population was an epileptic, and in 1881 in the Rhine Province 1 in every 1177. In Russia the proportion is variously stated as being 1 in 2000 by Kowalevsky,[7] and 1 in 1000 by Shoutelwort.[8]

[1] Burlureaux, quoted by Féré, *Les Epilepsies*, Paris, 1890, p. 246.

[2] Morselli, Art. 'Epilepsie,' Eulenberg's *Encyclopedia*, vol. vii. 1895.

[3] Binswanger, *Die Epilepsie*, 1899, pp. 173 and 174.

[4] Peterson, quoted by Spratling, *Epilepsy*, 1904, p. 47.

[5] Kolle, quoted by Spratling, *Epilepsy*, 1904, p. 46.

[6] Bircher, from *Reports of Charity Organisation Soc.*, 'The Epileptic and Crippled,' 1893.

[7] Kowalevsky, *National Association for Study of Epilepsy, U.S.A.*, 1901, p. 167.

[8] Shoutelwort, *ibid.* p. 167.

From the preceding figures, therefore, it may be gathered that in Europe, and the United States of America, the *approximate ratio of epileptics varies from* 1 *to* 3 *per* 1000 *of the general population.*

Epilepsy is not a common malady amongst the natives of India. From some figures which have been supplied to me,[1] a comparison has been made of the incidence of epilepsy amongst the European troops, the native troops, and the jail population respectively over a period of five years (1899 to 1903).

The table shows the frequency of epilepsy per 1000 individuals, based upon the averages of five years:

European troops,	1·6 per 1000	out of	63,848
Native troops,	0·4 „ 1000	„	124,456
Jail population (male and female),	1·2 „ 1000	„	113,016

From this it is obvious that the incidence of epilepsy amongst the British soldiers in India is four times as great as amongst the native soldiery, and that even in the jail population, where one would expect to find degeneracy well-marked and epilepsy frequent, the ratio per thousand is still below that of the white troops.

Epilepsy does not appear to be frequent in the Australian Commonwealth. From some figures taken from the Victorian Year Book of 1894, I found the average ratio per thousand of population in South Australia, Victoria, and Tasmania to be ·35.

According to Haoelburg[2] epilepsy is common in Brazil.

Spratling[3] gives some interesting facts upon the prevalence of epilepsy amongst the negroes in North America. "It is a generally expressed opinion that Insanity and Epilepsy are rapidly increasing amongst the negroes. They came out of their servitude, an inherently improved people, mentally and physically. . . . Under slavery, their habits, appetites, and tendencies to dissipate were largely regulated and controlled, but under freedom from slavery all their hurtful tendencies were allowed to run full riot." The result has been, he says,

[1] For the figures which are detailed in Appendix A, I am indebted to Surgeon-General Branfoot of the Indian Medical Service.

[2] Haoelburg, *National Association for Study of Epilepsy, U.S.A.*, 1901, p. 106.

[3] Spratling, *Epilepsy*, New York, 1904, p. 56.

that there are about thirteen times as many insane negroes under State care in Alabama as there were thirty years ago.

To permit of ready reference, the above facts and figures have been collected in tabular form as follows:

Table 1, *showing the ratio of Epileptics per* 1000 *of the general population from certain selected countries.*

Country.	Ratio per Mille.	Authority.	Date.
Prussia	2·9 per 1000	Binswanger	1895
Canton Aargau	2·5 „ 1000	Bircher	1893
France	2·5 „ 1000	Burlureaux	1890
United States	2 „ 1000	Peterson	1904
Montenegro	1·70 „ 1000	Miljanitsch	1877
Switzerland	1·5 „ 1000	Kolle	—
Italy	1·15 „ 1000	Morselli	1895
Mecklenburg Schwerin	1·17 „ 1000	Tigges	1882
Russia	1·0 „ 1000	Shoutelwort	1901
„	0·5 „ 1000	Kowalevsky	
Rhine Province	0·85 „ 1000	Pelman	1881
South Australia	0·43 „ 1000	Victorian Year Book	1894
Victoria	0·37 „ 1000		
Tasmania	0·25 „ 1000		
Indian Native Troops	0·4 „ 1000	Branfoot	1903

Urquhart (*Brit. Med. Journal*, 1905, 2, p. 1572) states that epilepsy is comparatively infrequent in Scotland, especially among the middle classes.

2. Relative Frequency in the Sexes.

The proportion of males and females who suffer from epilepsy has been variously stated by different writers, but the general impression gained from a perusal of the writings of authorities seems to favour its greater frequency in the female sex.

There are two ways by which the proportion between the sexes may be gauged with a fair degree of accuracy; first, by analysing a large number of cases such as attend for treatment at, or are inmates of, an Institution, or Hospital for Epilepsy; and secondly, by examining the statistical records of the Census Reports in cases of deaths from epilepsy.

The first method has been used in the compilation of the following table, which shows the statistical data given by several well-known writers on this subject:

Table 2, showing the percentage of males and females afflicted with Epilepsy, as stated by various authorities.

Name.	Total Cases.	Percentage. Males.	Percentage. Females.
Gowers[1] (England) - -	3,000	48	52
Tigges (Mecklenburg) - -	639	44	55
Echeverria[2] (U.S.A.) - -	306	42	58
Spratling[3] (U.S.A.) - -	1,582	60	40
Sinkler (U.S.A.) - - -	1,024	53	47
Binswanger[4] (Germany) -	—	61	38

From this it is evident that the first three writers, representing three separate countries, give a larger percentage of female than male epileptics, while the statisties of the three last show the converse.

The second method is to aseertain the total number of deaths from epilepsy in any given year and the proportion of males and females who annually succumb to this disease. However, in this method there is more seope for error, as many deaths arising from eelampsia, and other eonvulsive affections in adult life, may be ascribed to epilepsy.

Spratling gives the figures from the United States Census Bureau for a number of years, in all of which the male mortality was considerably in excess of the female.

I have taken the following figures from the Reports of the Registrar General, recording the deaths from epilepsy in England and Wales (exclusive of London) for the last three years of which the Reports are available.

In	1902	1593 males	1412	females.
„	1903	1598 „	1454	„
„	1904	1557 „	1357	„

From these figures it is obvious that from one to two hundred more males die annually from epilepsy than females.

As it would be of interest to eompare these figures with the number of deaths from insanity, the figures have been annexed

[1] Gowers, *Epilepsy*, 2nd edition, 1901, p. 15.

[2] Echeverria, *Epilepsy*, New York, 1870.

[3] Spratling, *Epilepsy*, New York, 1904, p. 56.

[4] Binswanger, *Die Epilepsie*, 1899, pp. 173 and 174.

from the Reports in this connection. The term Insanity excludes puerperal insanity and general paralysis of the insane.

In	1902	781	males	945 females.
„	1903	715	„	938 „
„	1904	866	„	1060 „

These figures show that a larger number of females die annually of the ordinary forms of insanity than males.

My own observations support the view that more males are afflicted with epilepsy than females, as shown in the annexed table:

Table 3, *shows the total number and percentage of the sexes in* 1000 *cases of Epilepsy.*

	Total.	Per cent.
Males - -	559	55·9
Females - -	441	44·1
Totals -	1,000	100

It has been maintained by Gowers,[1] that in the earlier years of life, when hereditary influences play a more important part in the production of epilepsy, the females considerably exceed the males in number, but that during adult life, when numerous accidental circumstances are at work in connection with the occupation and habits of men, the proportion is reversed. With a view to the confirmation, or otherwise, of this statement, the present series of cases has been divided into decennial periods, from which it is seen that there is no such marked variation at the several epochs as stated above.

Table 4, *showing the total number of males and females afflicted with Epilepsy according to decennial periods, from birth to* 70 *years.*

Age.	Total Males.	Total Females.
Birth to 10 -	180	133
11 „ 20 -	256	211
21 „ 30 -	68	54
31 „ 40 -	32	26
41 „ 50 -	16	10
51 „ 60 -	4	6
61 „ 70 -	3	1

[1] Gowers, *Epilepsy*, 2nd edition, 1901, p. 15.

The table shows that at all ages, with the insignificant exception of the decennium 51 to 60, the males exceed the females in number.

From the above facts it may be concluded that *more males than females are afflicted with epilepsy*, whether the figures are taken from 1000 consecutive cases of epilepsy seen at special Epileptic Institutions and Hospitals, or from the official death returns of the Registrar General. The observations upon the relative frequency of the sexes, according to the incidence of the disease in decennial periods, also afford confirmatory evidence, in that there is no special proclivity in the male sex during adult life, which is not also observed during the earlier years of infancy and childhood.

3. Age at onset.

The commencement of epilepsy is dated from the incidence of the first fit, a fact sometimes difficult to ascertain, as various causes tend to obscure the onset, of which the following may be stated to be the more important. First, fits may occur during sleep, and their existence remain undetected for many years, or even throughout life; secondly, the disease may have existed for some time in the form of "petit mal," or minor epilepsy, the significance of which is not recognised until a convulsive seizure calls attention to the true nature of the malady; and, thirdly, in many epileptics, the disease has commenced during the first few months of life, in the form of infantile convulsions, which, unless they continue in a chronic form, are not usually regarded as the true commencement of the disease.

Personal observations.—The statistics of most observers agree in showing that epilepsy is a disease of all ages, although there is a diminishing tendency to its occurrence as age advances. On the one hand there is no age which may be regarded as constantly, or consistently, exempt from the incidence of epileptic seizures. On the other, there are certain epochs during which the onset of epileptic fits is more common, as will be seen from a consideration of the subjoined table, showing the incidence of epilepsy yearly, from birth to seventy.

Table 5, *showing the total number of cases of Epilepsy with the age at commencement of the disease.*

Age.	No.	Age.	No.	Age.	No.
Under 1 year	100	At 22 years	16	At 43 years	3
At 2 years	22	23 „	9	44 „	2
3 „	17	24 „	6	45 „	2
4 „	14	25 „	14	46 „	3
5 „	17	26 „	12	47 „	2
6 „	18	27 „	9	48 „	3
7 „	40	28 „	14	50 „	1
8 „	19	29 „	7	51 „	2
9 „	25	30 „	11	53 „	1
10 „	41	31 „	7	54 „	1
11 „	37	32 „	5	55 „	1
12 „	68	33 „	11	56 „	1
13 „	40	34 „	5	57 „	1
14 „	71	35 „	6	58 „	1
15 „	65	36 „	6	59 „	1
16 „	44	37 „	7	60 „	1
17 „	48	38 „	1	63 „	2
18 „	36	39 „	3	67 „	1
19 „	30	40 „	7	70 „	1
20 „	28	41 „	4	Total, 1000 cases.	
21 „	24	42 „	7		

The following general observations may be drawn from a study of the preceding table:

1. The greatest number of cases of epilepsy, commencing in any single year, is to be found between birth and twelve months of age; the first year of life, therefore, is the commonest for the onset of the disease.

2. There is a rapid and extensive fall in the number of cases which commence between the first and the fourth years, from which latter age there is a slight increase up to the seventh.

3. A small decline then takes place about the eighth or ninth years, preliminary to the steady increase in the incidence of the disease, which reaches a second maximum between the ages of twelve and fifteen.

4. From this period onwards there is a steady, though not necessarily a progressive decrease, with, however, a slight temporary increase in the number of cases which commence at from twenty-five to twenty-eight years of age.

In order that a comparison may be more satisfactorily made with other statistical evidence upon the incidence of epileptic fits, the subjoined table has been drawn up to show the age at onset in quinquennial periods, and the number of each sex, with the percentage frequency in the several quinquennia.

Table 6, showing the numbers, sex, and percentage frequency of the cases in quinquennial periods from birth to seventy.

Age.	Total.	Males.	Females.	Percent.
Birth to 5 years - -	170	104	66	17
6 to 10 „ - -	143	76	67	14·3
11 to 15 „ - -	281	150	131	28·1
16 to 20 „ - -	186	106	80	18·6
21 to 25 „ - -	69	31	38	6·9
26 to 30 „ - -	53	37	16	5·3
31 to 35 „ - -	34	20	14	3·4
36 to 40 „ - -	24	12	12	2·4
41 to 45 „ - -	18	9	9	1·8
46 to 50 „ - -	8	7	1	·8
51 to 55 „ - -	5	2	3	·5
56 to 60 „ - -	5	2	3	·5
61 to 70 „ - -	4	3	1	·4
	1000	559	441	100

In rather more than one quarter of the total number (28·1 per cent.) the disease commences between eleven and fifteen years of age, and in very nearly one half (46·7 per cent.) during the decennium eleven to twenty years; while more than three-quarters of all the cases (78 per cent.) commence between birth and the age of twenty years.

The large proportion of cases commencing during the first year of life, is a point of great importance, as the above list embraces only cases of genuine epilepsy, and not instances merely of infantile convulsions.

These facts are in general agreement with the figures given by Hasse, Gowers, Spratling, and other writers, who have handled large numbers of cases from which to draw their conclusions.

The table also shows, what has been noted on a previous page, that at all ages, with a few insignificant exceptions, the males exceed the females in number, a fact which is at variance with

the most recent figures of Gowers, whose statistics, in other respects, are uniformly in agreement with those presented here. Gowers states, that at each of the maximum periods the excess of females is great; that during the later period of childhood the numbers are nearly equal, and that the excess of females lessens after puberty, and ceases by middle life.

It is preferable, however, to subdivide the age incidence into three unequal periods, as it is by this means that a clearer conception is obtained of the powerful influence which development and growth play in the causation of epilepsy.

The annexed table shows the numerical and percentage frequency of the onset of epileptic fits according to the three epochs of childhood, puberty and adolescence, and adult life:

Table 7, showing the total numbers and percentage frequency of the cases of epilepsy, classified according to the three epochs described in the text.

Age.	Total.	Percentage.	Percentage with similar Heredity.
Birth to 9 years - - -	272	27·2	6·0
10 to 23 „ - - -	557	55·7	11·9
24 to 70 „ - - -	171	17·1	2·2

The *first epoch* is from birth to eight or nine years of age, a period corresponding to infancy and childhood. This epoch may be subdivided into the periods of rapid brain growth occurring during the first year of life, and the more slow but progressive development of childhood. The percentage of cases commencing during this epoch is 27·2, of which no less than 10 per cent. arise during the first twelve months of life. Reference to table 5 will show a considerable increase at seven years of age. The explanation of this rise is probably to be found in the temporary irritation induced by the eruption of the second dentition.

The *second epoch* is from ten to the ages of twenty-two or twenty-three. This embraces the rise and full development of the reproductive functions. It is during this period that the onset of epilepsy attains its greatest numerical frequency, no less than 54·8 per cent. of all cases commencing during this epoch. The age of maximum incidence is seen to be fourteen, which corresponds to the onset of puberty in the majority of individuals.

This epoch merges gradually into *the third*, which includes the remainder of life; but at or about the ages of twenty-five to twenty-eight there is observed a slight, and temporary recrudescence in the number. There is no statistical evidence that the climacteric is associated with any special tendency to the development of epileptic fits.

While referring to the epochal epilepsies, there are two points deserving of mention. The first is the not uncommon spontaneous arrest of fits, which is observed during childhood, usually from the ages of three, four or five, up to the commencement of puberty. This period corresponds to the later years of the first epoch, during which the incidence of epilepsy is numerically small.

The second point is the less commonly observed spontaneous arrest of fits during the development of the reproductive functions, a period corresponding to the whole of the second epoch, during which the onset of the disease attains its maximum frequency. In the cases of this character which were observed, fits, commencing in infancy, ceased at twelve to sixteen years of age, to recur after periods varying from five to sixteen years. These cases eventually became confirmed epileptics, but further reference is made to them when remissions in epilepsy are more fully considered (p. 114).

Mean age at onset. With the facts at our disposal, it is possible to ascertain the mean age at which the disease usually commences, a point of some importance, as it may be shown that the average age at commencement varies slightly in the two sexes. Moreover, where a hereditary history was obtained, the mean age at onset was somewhat in advance of those cases which presented no heredity.

These points may best be demonstrated by reference to the subjoined table, which shows the mean, or average age, at onset in the total cases, in the male and female cases, and in the cases with a hereditary history of epilepsy:

Table 8, *showing the mean age at onset in the sexes, and in hereditary cases.*

Total cases - -	1000,	mean age	14·8	years	at onset.
Males - - -	559,	„	13·7	„	„
Females - - -	441,	„	15·5	„	„
With similar heredity	201,	„	13·3	„	„

It may be concluded from this table that the mean average

age of all cases is coincidental with the onset of the reproductive functions at puberty; but that, somewhat unexpectedly, the onset of the disease in young women is later by about two years than in the male sex. The existence of a family history of epilepsy determines a slight advance in the average age at onset.[1]

These figures show the onset of epilepsy to be somewhat later than that described by Doran,[2] who has studied the influence of the several forms of heredity in determining the average age at the commencement of the disease.

The following are Doran's figures:

Average age at commencement - -	12·36	years.
,, ,, without Heredity - -	13·36	,,
,, ,, with Neuropathic heredity	11·2	,,
,, ,, with Alcoholic heredity -	10·8	,,
,, ,, with Heredity to Insanity	10·8	,,
,, ,, with Heredity to Epilepsy	10·17	,,
,, ,, with Heredity to Epilepsy and Alcoholism	9·9	,,

4. Heredity.

General considerations. Many difficulties stand in the way of properly discussing the true bearing of heredity in the causation of epilepsy, but there would seem to be less likelihood of error in this direction, than in the closely related subject of insanity. It is generally accepted that epilepties exhibit in their structural formation and mental characteristics, definite and well-marked signs of an inherited neuropathic taint, the so-called signs or "stigmata" of degeneration. These peculiarities are less apparent in the insane, in whom environment, personal idiosyncrasies, and individual temperament play almost as important a part as the hereditary factor. On the other hand, in the dementias of childhood—idiocy and imbecility—the anatomical side of the neuropathic disposition is more fully seen than even in epilepsy.

It is therefore necessary to ascertain whether, and how far,

[1] Reynolds (*Epilepsy*, London, 1861) gives the following figures, which differ somewhat from those recorded here: Mean age (with heredity)—males, 15 years; females, 12 years. Without heredity—males, 16 years: females, 13 years.

[2] Doran, *American Journal of Insanity*, 1903.

neuropathic tendencies are demonstrable in the ancestors of epileptics, or in the family histories. In order to answer this question one is led to the consideration of the hereditary history, and the factors which determine the onset of epileptic attacks in predisposed individuals.

It may at the outset be stated in general terms that neuropathic hereditary tendencies are either of a similar, or dissimilar character; the former term being applied when the disease in the offspring is of the same character as shown in the parent, viz., epilepsy; the latter when the parental predisposition manifests itself in some other disease of the nervous system, such as alcoholism or insanity.

The influence of heredity in the causation of epilepsy has been fully studied by all writers on the subject, and as the statistical method has been the one necessarily adopted, the results have varied within considerable limits. The causes of these discrepancies are traceable to numerous factors, but the principal ones may be stated to be: first, the difficulty in obtaining precise information upon the family antecedents from parents or relatives, mainly through ignorance, which is more particularly seen amongst hospital cases; and secondly, from the inclusion by many writers, amongst hereditary factors, of maladies which do not stand in any direct causal relation to epilepsy, but are merely thrown in occasional connection with it, such as, tuberculosis, gout, and rheumatism. Further causes for these differences may also be found in the limitation of enquiries to parental afflictions, without reference to the existence of collateral heredity, in the concentration of attention on a particular hereditary malady, and in the personal ability of the investigator in the elicitation of facts.

It would therefore be of interest at the outset of the investigation into the influence of hereditary factors, to ascertain how far the observations of some of the recent writers on epilepsy throw light on this aspect of the disease. A table has therefore been constructed to show the percentage frequency of hereditary factors from the statistical researches of several recent writers:

[Table 9

Table 9, *showing the percentage of Epileptics with an hereditary history, as stated by several recent writers.*

Author.	Total Cases.	Percentage with Hereditary History.
Echeverria[1] - -	306	25·0
R. Reynolds[2] - -	38	31·0
Moreau[3] - - -	364	32·0
Aronsohn[4] - - -	508	32·0
O. Berger[5] - - -	71	32·3
Binswanger[6] - -	150	36·3
Gowers[7] - - -	2400	40·0
Spratling[8] - -	1070	56·0
Déjérine[9] - - -	350	66·8

This table shows very clearly how considerable the discrepancies may be between authorities on the subject—a discrepancy varying from 25 to 66 per cent. as the expression of the hereditary element in the causation of epilepsy. Some of these observers have included in their statistics nervous maladies, which do not stand in any direct causal or hereditary relationship, such as apoplexy, neuralgia, locomotor ataxy, and general paralysis of the insane. Hence, in order to ascertain how far definitely neuropathic maladies play a part in the causation of epilepsy, the following table has been constructed to show the percentage frequency of the three main hereditary factors in the ancestral history of epileptics, viz., epilepsy, insanity, and parental alcoholism:

Table 10, *showing the percentage frequency of Epilepsy, Insanity, and Alcoholism as hereditary factors in the causation of Epilepsy.*

	Déjérine.	Binswanger.	Spratling.	Doran.[10]
Epilepsy - -	21·2 %	11 %	16 %	19·3 %
Insanity - -	16·8 %	29·6 %	7 %	7·9 %
Alcoholism[11] -	51·6 %	22 %	14 %	21·6 %

[1] Echeverria, *Epilepsy*, New York, 1871. [2] Reynolds, *Epilepsy*, London, 1861.
[3] Moreau, quoted by Binswanger, *op. cit.* p. 77.
[4] Aronsohn, *Neurol. Centralb.* 1894, p. 631.
[5] Berger, quoted by Binswanger, *op. cit.* p. 79.
[6] Binswanger, *Die Epilepsie*, 1899, p. 82.
[7] Gowers, *Epilepsy*, 2nd edit. 1901, p. 7. [8] Spratling, *Epilepsy*, 1904, p. 64.
[9] Déjérine, *Hérédité dans les Mal. du Syst. Nerv.*, 1886.
[10] Doran, *American Journal of Insanity*, 1903, p. 61.
[11] As bearing directly on the frequency of alcoholism as a hereditary antecedent of epilepsy, it may be added that Féré[12] found this predisposition in 38·3 % of his cases, and J. Voisin[13] in 31 %.
[12] Féré, *Les Epilepsies*, Paris, 1890. [13] Voisin, *L'Epilepsie*, Paris, 1897.

The outstanding feature of this table is the enormous percentage frequency of alcoholism as noted by the French observers, an antecedent disposition far outweighing the combined percentages of epilepsy and insanity, according to Déjérine's figures. The German figures, on the other hand, show the predominating influence of insanity, whilst the American give almost equal prominence to epilepsy and alcoholism as leading hereditary factors in the causation of the disease. Amongst English writers, Gowers expresses the opinion that parental intemperance is probably due in many cases to a neuropathic disposition, but is seldom to be trusted as its evidence. He gives no figures bearing upon the relative frequency of the three factors above mentioned.

In the heredity of epilepsy three main influences have therefore to be considered, viz., epilepsy, insanity, and parental alcoholism.

Personal observations. In the series of cases of epilepsy collected for the purposes of this research, enquiry was directed primarily to the influence of epilepsy itself, and to insanity and alcoholism in a secondary manner. As it would appear to be of importance to ascertain the influence of collateral as well as of direct heredity, information was also sought upon this point.

The ascertained facts bearing upon heredity are shown in the following table:

Table 11, *showing the total number of cases and percentage frequency of the hereditary factors in* 676 *Epileptics, in whom the history was as far as possible investigated.*

Disease.	Total Cases.	Percentage.
Epilepsy - - -	252	37·2
Insanity - - -	37	5·4
Alcoholism - - -	21	3·1
Other nervous disorders[1]	36	5·3
No known heredity -	330	49·0
Totals - -	676	100·0

From this table it is evident that *the most common predispos-*

[1] Under this heading are included, chorea, "nervousness," migraine and paroxysmal headache, suicide and deaf-mutism.

ing cause of epilepsy is ancestral epilepsy; insanity and parental alcoholism exerting only a minor and secondary influence. It is therefore clear that a similar heredity is the main hereditary factor found in the family histories of these patients.

Some of the older writers believed this to be extremely rare, but the statistical information acquired by the most recent authorities points to its importance, although its relative influence as the main element varies in the statistics from different countries.

In order to show the relative frequency of the several hereditary factors from amongst the two groups of patients used in this investigation, viz., those seen in private in contra-distinction to those seen in hospital practice, a further table has been constructed giving the percentage frequency of the chief hereditary factors:

Table 12, *showing the percentage frequency of Epilepsy, Insanity, and Alcoholism as hereditary factors, in hospital and private practice respectively.*

	Epilepsy.	Insanity.	Alcoholism.	Other.	Totals.
Hospital - -	35·9 %	4·2 %	3·0 %	5·4 %	48·5 %
Private - -	46·7 %	13·0 %	3·2 %	4·3 %	67·2 %
Total Cases	37·2 %	5·4 %	3·1 %	5·3 %	51·0 %

It is obvious from this table that a considerably higher percentage of cases from private practice present a hereditary history of neuropathic maladies than the hospital cases, viz., 67 per cent., of which 46 per cent. were to epilepsy alone. In only 35·9 per cent. of the hospital patients, on the other hand, was there obtained evidence of family epilepsy.

The most striking feature of the tables is the predominating influence of ancestral epilepsy as the most important factor in the hereditary history of this disease—a further corroboration of the view held by most English writers on this subject. As already shown, this is in opposition to the expressed opinion of some French authors, whose statistics place parental alcoholism in the foremost rank.

The rôle of insanity and alcoholism in the family histories of epileptics, though secondary, ought not to be disregarded; of lesser importance, and indicating rather the general trend of

the psychopathic hereditary disposition, such other affections as chorea, "nervousness," paroxysmal headache, suicide, and deaf-mutism ought not to be overlooked.

Collateral heredity. The hereditary transmission of epilepsy may be direct, the predisposition being acquired from one or other parent; on the other hand, the family hereditary tendency may reveal itself in collateral relatives. In this way, a history of epilepsy may be obtained, not in the parents, but in the uncles, aunts, or cousins of the persons so afflicted. Although no obvious hereditary degenerative psychosis may be found in the parents, two or more members of the same family, such as a brother or a sister, may suffer from epileptic fits. A more careful enquiry into the parental history in such cases will usually bring out some evidence of a hereditary neurosis, as, for instance, periodic headaches, "bilious" attacks, migraine, or chorea. In a few cases there was evidence of unsuspected, or unrecognised, epilepsy.

A summary of the numerical frequency of the disease found amongst the relatives of those suffering from epileptic fits from 676 cases is given in the annexed table:

	Cases.
Epilepsy in the father	50
,, ,, father's collaterals	35
,, ,, paternal grandparents	16
Total	101
Epilepsy in the mother	33
,, ,, mother's collaterals	42
,, ,, maternal grandparents	14
Total	89
Epilepsy in members of the same family, brothers or sisters	62
Total cases examined	676

From this summary it may be stated that, in a general way, the disease is more frequently transmitted from the father's, than from the mother's side. The number of cases, giving a family history of insanity, showed an equality as regards the paternal and maternal transmission; while of those in which a history of parental alcoholism was given, a somewhat larger number presented a history of inebriety on the father's side.

In relation to this aspect of heredity, there is the close connection which is noted between epilepsy in the parent, and infantile convulsions in the offspring.

This subject has been studied in detail by Echeverria,[1] who showed that out of five hundred and fifty-three children, born of one hundred and thirty-six epileptic parents, one hundred and ninety-five, or 35 per cent., died from convulsions in infancy.

In the offspring of an epileptic parent, on the other hand, fits may, or may not, appear. There would seem to be no guide to the solution of this question. In the present series of cases there were illustrations of both; but without desiring to draw conclusions from a small number of instances, the following observation was made; that in the cases in whose family there was no known hereditary history of epilepsy, fits had not appeared in the children; but that in the cases in which the disease was obviously hereditary, fits or convulsions were present in one or more of the offspring.

A minor point of some interest is the fact that the child may develop fits at an earlier age than the parent; thus a father had epilepsy at 42 years, his daughter at 21; another had a fit towards the end of his life, his daughter at 35 years; other similar observations are common.

Although epilepsy and insanity are the two main elements of the psychopathic hereditary degeneration, the existence in the family history of hysteria, chorea, the drug habit, migraine and paroxysmal headache, are important, not so much from any direct bearing which they may have upon the development of epilepsy, but as indications, to some extent, of the neuropathic tendencies of a family. We find such disorders not uncommon in the family and personal histories of epileptics, but it is difficult to prove that their occurrence is specially frequent.

Frequency of heredity. Epilepsy is a disease in which heredity plays an important part, but its influence is not limited to the earlier years of infancy and puberty, as will be seen from a consideration of the following table:

[Table 13

[1] Echeverria, *Amer. Journal of Insanity*, 1880.

Table 13, *showing the percentage frequency of an epileptic heredity during the first year of life, and in decennial periods thereafter.*

Age.	Total Cases.	Percentage with Epileptic Heredity.
Under 1 year - - -	100	22·0
2 to 10 years - - -	213	22·0
11 to 20 „ - - -	467	22·0
21 to 30 „ - - -	122	15·5
31 to 40 „ - - -	58	10·3
41 to 50 „ - - -	26	8·0
51 to 60 „ - - -	10	20·0
61 to 70 „ - - -	4	0·0

This table shows that the pereentage frequency of hereditary epilepsy is uniform throughout the first twenty years, during which period, abont 78 per eent. of all eases arise; but that during the later decennia, although the freqneney of heredity is lessened, the total number of eases is also materially reduced. Up to twenty an hereditary history of epilepsy was obtained in 22 per eent., and from twenty-one to seventy it was present in 13 per cent.

As pointed out by Gowers, it is less easy to obtain an hereditary history as life advanees, and the preceding generation passes out of the reach of questions. But notwithstanding this faet, the percentage of hereditary cases between 41 and 70 years of age amounts to 10 per eent. out of a total of 40 eases.

These figures largely eorroborate those given by Gowers, and show that the stress and strain of life, and the ehanging physiological eonditions assoeiated with inereasing years, do not to any great extent replace the hereditary influences in the eausation of epilepsy. Henee it is found that in the so-ealled Senile Epilepsy, although arterio-selerotie changes may play a part in its development, the hereditary factor is still prominent and active.

5. The Signs or "Stigmata" of Degeneration.

The indications of a neuro- or psyehopathie hereditary tendency are shown in a variety of ways, by what are known as the signs, or "stigmata" of degeneration. These may be defined as structural, and functional, deviations from the normal, and

point to the existence of a latent neuropathic disposition, which may exert a potent influence upon the causation, type, course, and treatment of nervous and mental diseases.

"Stigmata" of degeneration have been divided for descriptive purposes into the anatomical and the psychical; either, or both, of which, in greater or less degree, are found in the subjects of epilepsy.

They are of immense value as an index of the intensity, or degree, of the hereditary degenerative predisposition. In the most pronounced forms of mental deficiency, such as are seen in imbeciles, idiots, and congenital epileptic dements, anatomical variations from the normal are common and often of a pronounced type. Such abnormalities are exemplified in microcephaly and other cranial deformities; in prognathism and cranio-facial asymmetry; in defective development of the hard palate; in irregularities and displacements of the teeth; in malformed limbs, and in badly shaped ears.

In the slighter forms of neuroses, such as neurasthenia and migraine, and in the lesser psychoses, simple melancholia, and senile insanity, the neuropathic stigmata are less frequent and less pronounced than in the more exaggerated conditions above mentioned.

In epilepsy, which from many points of view may be regarded as occupying a position midway between the less and the severe degenerative psychoses, stigmata of degeneration are present; these have received considerable attention more especially at the hands of C. Féré.[1] In addition to the cranial asymmetry, which Lasègue considered to be constant in true epilepsy, Féré has called attention to the frequency of cranio-facial asymmetry amongst epileptics. It is not absolutely characteristic of the disease, as it is found in a certain small percentage of healthy individuals.

Among other features of a degenerative character, the configuration of the hard palate deserves special attention. This has been the subject of much investigation and discussion, and its study and relation to the several degenerative psychoses is associated with the names, more especially in this country, of Langdon-Down and Clouston.[2]

Deformities of the ear have for long been regarded as important

[1] Féré, *Les Epilepsies*, 1890, p. 381.

[2] Clouston, *The Neuroses of Development*, Edinburgh, 1891.

structural stigmata. Abnormal implantation of the teeth in connection with epilepsy and the developmental neuroses has been the subject of careful investigation by Sollier[1] and Talbot.[2] In addition to the above mentioned stigmata, Féré has also drawn attention to, and described in detail various asymmetries of the thorax, pelvis, and limbs.

In whatever form they are met, it has to be borne in mind that stigmata of degeneration are structural or psychical deviations from the normal, occurring during the period of growth and development of the central nervous apparatus, in those who are the subjects of a hereditary degenerative predisposition.

Of the two quinquennial periods in which the onset of epilepsy is more common, that from birth to five years of age is the most fruitful in the production of stigmata. It is during this period that the growth and development of the brain is most active; it is during this period also that the other hereditary degenerative neuroses and psychoses appear, such as stammering, backwardness in walking and talking, delayed dentition, deaf-mutism, and some forms of idiocy and imbecility (Clouston).

Personal observations. The following are described as amongst the more commonly observed structural stigmata found in epilepties. Two hundred cases were examined with a view to determine the frequency and variety of such signs of degeneration.

1. *Facial deformity and asymmetry.* Under this heading are included, inequalities of the two sides of the face, in whole or in part; irregularities of the nose, not arising from traumatic causes; prognathism and feeble, or arrested, development of the lower jaw. Arrested development of the upper jaw is indicated in the state of the hard palate.

According to the investigations herein detailed, facial asymmetry is more common amongst female than male epilepties: moreover it rarely occurs alone (in only 14 per cent.), being associated with other well-marked structural stigmata. Forty-two per cent. of the cases showed facial asymmetry, nasal deformity, and defective development of the lower jaw.

2. *Deformities of the hard palate.* Abnormal development

[1] Sollier, (Mde.), *De l'Etat de la Dentition*, Paris, 1887.

[2] Talbot, *Irregularities of the Teeth*, Philadelphia, 1901.

of the hard palate has long been recognised as one of the most constant of the degenerative stigmata. Various types of abnormal palate have been described by different observers, but I have adopted Clouston's[1] classification, as being at once simple and comprehensive. This authority has described three varieties: (*a*) the normal, or "typical," palate, which is an arch-shaped structure, with a low, regular, and wide dome; (*b*) the "neurotic" palate, which is higher than the normal, somewhat narrower, but has a fair dome-shaped arch. It is the palate commonly seen in persons of a nervous temperament, and is not infrequently met with in neurasthenies, sufferers from migraine, depression, and dipsomania; (*c*) the "deformed" palate, which presents various abnormal shapes, is very high, narrow, keel-shaped, and somewhat irregular, and its roof has a V-shaped, or saddle-like, appearance. Further the *torus palatinus* is a mesial prominence along the palatal suture, and occupies the whole or part of the palatal roof. Cases presenting this abnormality have been placed in a fourth subdivision.

Like facial asymmetry, palatal deformity as a neuropathic stigma, does not usually exist alone, only 18·5 per cent. of the present series showing this deformity unassociated with other obvious evidence of structural degeneration.

The subjoined table shows the percentage frequency of the four types of palate in two hundred epileptics:

Normal, - - -	54	per cent.
Neurotic, - - -	26	„
Deformed, - - -	13·5	„
Torus palatinus - -	2	„

This gives a total of 41·5 per cent. with palatal stigmata of degeneration.

3. *Dental anomalies* as evidence of a neuropathic hereditary predisposition, are found, according to Sollier,[2] in about 90 per cent. of the feeble-minded, with or without epilepsy. The characteristic changes are seen chiefly in connection with the second dentition. The observations of Sollier, from whom these statements are quoted, afford proof of the following as characteristic stigmata in dentition:

[1] Clouston, *The Neuroses of Development*, Edinb., 1891.

[2] Mde. Sollier, *De l'Etat de la Dentition*, Paris, 1887.

(*a*) Retardation in the eruption of the second dentition (25 per cent.).

(*b*) Abnormal forms of the teeth, including "dwarfism" and "giantism."

(*c*) Abnormal implantation of the teeth, with which is associated abnormal direction chiefly of the incisors and the canines.

(*d*) Longitudinal striation.

(*e*) Anomalies of the dental arches, more especially an inequality of the two halves, particularly of the upper jaw.

Faulty enamelling of the teeth is frequent in epileptics, and in defectives as a class; while in both the teeth are notoriously prone to early caries.

4. *Deformities of the ears.* Although the relationship between the shape of the hard palate, the formation of the base of the skull, and the development of the brain would appear to be fairly clear and generally recognised, it is less easy to understand the connection between the shape of the ears and the degree of brain growth; yet the external ear presents a constant and fruitful field for the study of neuropathic stigmata. Abnormalities of the ears have been extensively studied by numerous observers, as many as twenty-two varieties of aural deformity having been described (Peterson).[1]

For the present research the following subdivisions have been adopted:

(*a*) Normal ears.

(*b*) Abnormally shaped ears. This includes ugly ears; ears without, or with adherent, lobules; excessive conchoidal development; abnormal thinning of the margin; deformities of the antihelix; the Morel ear,[2] and other atypical structural appearances.

(*c*) Abnormal size. Ears that are too large, too small, or too long.

(*d*) Asymmetrical ears—the Blainville ear.

As with the other stigmata, an abnormal development of the ear is rarely seen alone, only 7·5 per cent. of the cases showing such a solitary stigma.

[1] Peterson, *Nervous and Mental Diseases*, Church and Peterson, New York, 1904.

[2] The *Morel ear* is that form of auricle marked by abnormal development of the helix, antihelix, fossa scaphoidea and aura furcata, so that the folds of the ear seem obliterated, and the ear is smooth, larger than usual, often prominent, and with a thin edge (Peterson).

The subjoined table gives the percentage frequency of the different forms of ear in 200 epileptics:

Normal ears,	- -	65 per cent.
Abnormal shape,	- -	10 „
Abnormal size,	- -	8 „
Asymmetrical ears,	-	5 „

or a total of 23 per cent. with aural stigmata.

It does not seem possible to connect any particular degree of mental impairment with the different forms of aural abnormality. It may, however, be stated in general terms, that the more pronounced the deformity, such as is seen in the Morel ear, in unusual conchoidal development of the auricle, and in abnormally large and prominent ears, the greater is the degree of mental enfeeblement.

The following table has been constructed to show at a glance the relative frequency and percentage of the three chief forms of structural stigmata described above in 200 epileptics:

Table 14, *showing the percentage frequency of stigmata of degeneration in* 200 *Epileptics.*

	Facial asymmetry.	Abnormal palates.	Abnormal ears.
In 200 Epileptics	42 %	41·5 %	23 %

5. Various other anatomical evidences of a hereditary degenerative character have been alluded to by writers on this subject, but it seems scarcely necessary to do more than mention some of them; such are, chromatic asymmetry of the iris, congenital cataracts; and anomalies of the limbs, such as polydactyly, syndactyly, and disproportionate length of the arms.

6. *Cerebral Stigmata.* Stigmata of degeneration, indicating the existence of a constitution hereditarily predisposed to mental instability and convulsions, are not limited to the anatomical phenomena just described. Recent researches upon the finer anatomy of the cerebral cortex have demonstrated peculiarities, which some authorities are inclined to regard as cerebral anatomical stigmata, and by aid of which the hereditarily or congenitally defective brain may be recognised. These are described more fully in the chapter upon the pathological

anatomy of epilepsy (p. 167), but are briefly referred to here as cerebral stigmata of degeneration.

(*a*) The nerve cells, occupying the outer layers of the cerebral cortex are materially reduced in number, but especially in the second and third layers, in which latter position the small pyramidal cells are affected. The diminution in number of the cells is an approximate guide to the degree of imbecility; while in appearance the cells are stunted and with few processes, the cytoplasm stains deeply and the nucleus is small and homogeneous.

(*b*) The large pyramidal or Betz cells show displacement of the nucleus, which is generally clear and well defined. The body of the cell is also large and its centre is occupied by fine, closely-set granules. This type of cell is stated by Lugaro[1] to be the normal type in some of the lower vertebrates. A somewhat similar appearance is also found in the large cells of the anterior horns of the spinal cord in idiots. Dr. John Turner found this embryonic type of cell in 77 per cent. of his cases of epilepsy. (Figs. 1 and 2.)

(*c*) A persistence of nerve cells in the white matter and the outer layers of the cortex (Roncoroni).[2] This is a normal feature of the brains of the lower vertebrates, new-born infants, and imbeciles at all ages; but in normal adults these cells have almost entirely disappeared.

(*d*) An alteration in the arrangement of the tangential, or association, fibres whereby a space, free of nerve fibres, is found in the inner half of the first cortical layer (Kaes).[3]

It is not unlikely that these appearances of the nerve cells described as 'embryonal,' may indicate an imperfectly developed nervous system, acquired by heredity, and prone to be influenced by accidental or exciting causes towards the development of convulsions.

7. In addition to the preceding structural abnormalities, which have been studied in detail owing to their greater frequency and importance, various others of a pathological character have been observed and described by writers upon epilepsy. We owe to Féré, more particularly, amongst recent authorities, detailed accounts of several stigmata of this type.

Astigmatism, was found by Féré to be present 72 times in 100 epileptics. The relation between errors of refraction and epileptic

[1] Lugaro, *Riv. Speriment di Freniatria*, 1902.
[2] Roncoroni, *Arch. di Psichiatria*, 1896. [3] Kaes, *Neurol. Centralblatt*, 1904.

seizures is important, in view of a causal connection which is believed to exist by some writers, and which has formed the basis of certain forms of treatment to be referred to in a subsequent chapter. Work Dodd[1] found simple hypermetropia less frequent in epilepties than in the apparently normal; but astigmatism, more especially the compound hypermetropic form, was much more common in epilepties than in normal persons. My observations on this subject have been confined to the detection of high errors of refraction, which were found to be present in 7 out of 200 cases, giving a percentage of 3·5. High myopia (beyond − 5 diopters) was observed in 5 cases; and high hypermetropia (beyond + 6 diopters) was found in 2 cases.

Stammering is a not uncommon degenerative stigma in epilepties. It was present in ten out of two hundred cases, or 5 per cent. of the present series.

Reference to the co-existence of *the 'ties'* with epilepsy is made upon a later page; but considering the essentially degenerative nature of this affection, their infrequency in epilepties is worthy of a passing note.

8. The *psychical stigmata* are found essentially in the peculiarities of the epileptic temperament, which is more fully discussed in the chapter upon the mental phenomena found in epilepsy; but to this may be added, as evidence of a degenerative heredity such abnormalities as sexual perversions and debility, eccentricity, precocity, abnormal feebleness or perversion of memory, and obsessions.

Clinical relations of the stigmata of degeneration.

1. *Hereditary disposition to epilepsy and insanity.* Owing to the difficulty in obtaining precise and satisfactory information upon the hereditary history in many of the cases, it has not been found possible to draw up so complete a tabular representation as has been done in connection with other matters; but it has been observed, that amongst those in whom an hereditary tendency was transmitted directly from one or other parent, the structural abnormality was more pronounced than in those who gave merely a collateral hereditary history. There were, on the other hand, not a few cases in which direct parental heredity was unassociated with any obvious anatomical evidence of developmental deformity.

The subjoined *table* (15) *shows the numerical and percentage*

[1] *Brain*, vol. 16, p. 534.

frequency of a hereditary family history, in two hundred epileptics, and its relation to the presence of structural stigmata:

Heredity.	Frequency.	Percentage.	Percentage with Stigmata.
Epilepsy on father's side -	18 cases	9·0	7·5
Epilepsy on mother's side -	28 „	14·0	8·0
Brothers or sisters epileptic -	9 „	4·5	3·0
Insanity - - - - -	11 „	5·5	4·0
Parental alcoholism - -	4 „	2·0	1·5
No known heredity - -	130 „	65·0	42·0
Totals - - -	200 cases	100·0	66·5

This table is instructive in so far as it shows that, of 200 cases of epilepsy, 42 per cent. presented well-marked evidences of structural stigmata, although no hereditary neuropathic history could be obtained; while of those in whom such a history was known, only 24 per cent. showed stigmata. It is therefore obvious that the absence of a family neuropathic history is of little account, in face of the well-marked structural signs of an inherited degenerative disposition, which many of these cases presented. Moreover, it is clear, that if the family history could have been probed more deeply, a larger percentage of those with stigmata of degeneration, would have made mention of some inherited degenerative psychosis.

2. *Age at Onset of the Convulsions.*—The following table was constructed with a view to ascertain whether the presence of stigmata of degeneration stands in any relationship to the age at the onset of the disease.

The table shows the total number of cases examined, and the frequency and percentage of those presenting stigmata of degeneration, according to the age at the onset of the seizures, arranged in quinquennial periods under twenty years:

Table 16 *showing the age at onset of Epilepsy and the percentage frequency of cases with stigmata of degeneration.*

Age at Onset.	Total Cases.	With Stigmata.	Percentage Frequency of Stigmata.
Birth to 5 -	46	32	69·5
6 „ 10 -	27	15	55·5
11 „ 15 -	69	45	65·2
16 „ 20 -	33	20	60·0
Over 20 -	25	16	64·0
Totals -	200	128	

It is therefore apparent that of the two quinquennial periods —birth to five, and eleven to fifteen years of age—in which the onset of epilepsy is more common, the former is the more fruitful in the production of structural neuropathic stigmata.

It will be shown on a later page (Chapter VI., p. 147), that the highest percentage of those who show profound mental impairment, is found amongst the cases of epilepsy which commence before the fifth year of age; and, in consequence, epilepsy commencing in infancy and childhood is the least amenable to treatment, and the most prone to become confirmed.

General Conclusions.

It has been shown in the preceding pages that epilepsy is a prevalent disease, and although its relative frequency varies according to the obtainable statistics in different countries, it may be stated that, on an average, two persons in every 1000 of the population are epileptic.

Although the statistics are also at variance as to whether epilepsy is more common in men than in women, there is ample evidence at hand to show, that the former are probably more subject to the disease than the latter; and further, that this greater frequency in the male sex does not necessarily depend upon the greater stress and strain to which the male is exposed in adult life, but is a feature of the disease from infancy to old age.

The great predisposing cause of epilepsy lies in a neuropathic predisposition, which manifests itself in various psychical, pathological, and structural peculiarities. Although a neuropathic family history was only obtainable in about 50 per cent. of the cases of epilepsy, the absence of such history does not necessarily imply its non-existence, in view of the presence of well-marked stigmata of degeneration in many of the cases.

It has also been pointed out, and evidence has been deduced from statistics to show, that the disease may make its appearance at any age, without ascertainable exciting cause. As fully three-quarters of the cases of epilepsy arise under twenty-one years of age, it is seen to be essentially a disease of the springtime of life, a period during which the development and growth of the central nervous system, and the maturation of the organs of reproduction play an important part. It is during

these years that the nervous instability acquired by heredity is most effective; and it is during this period of life that causes altogether insignificant, or insufficient in stable nervous systems, may light up the tendency to convulsions, which primarily characterise this malady.

It is therefore obvious that in the majority of cases of epilepsy, no external exciting cause of the disease is necessary. Many conjectural explanations are given by the patient or his friends, more particularly because it is inexplicable to them how so awe-inspiring and gruesome a malady may arise without apparent reason. Hence we find mentioned as ascribed causes, trivial head injuries, long forgotten until the first fit takes place, overwork at school, a so-called "sunstroke," and numerous other incidents, while the essential cause and real explanation is to be found in the rapid brain growth during the first few years of life, the onset of puberty, and the full development of the reproductive organs, in persons anatomically predisposed by heredity to nervous instability and convulsions.

It has also been shown that structural stigmata of degeneration, more particularly of the face, teeth, palate and ears, are frequent phenomena in the subjects of epilepsy, and that their presence is of great importance in determining, not only the degree of the inherited predisposition, but also the severity of the disease. Such signs of degeneration are more frequent when the disease begins early in life—under ten years of age, and form an important guide in the subsequent study of the malady, whether from the point of view of diagnosis, prognosis, or treatment.

CHAPTER III.

ETIOLOGY OF EPILEPSY (*continued*).

Determining causes of epilepsy—Reproductive functions—Sleep—Infantile convulsions—Infective disorders—Psychical causes—Trauma—Syphilis—Infantile hemiplegia—Organic epilepsy—Miscellaneous causes—Summary.

DETERMINING CAUSES OF EPILEPTIC ATTACKS.

It has just been shown that the essential cause of epilepsy is found in an inherited neuropathic disposition, which shows itself in the majority of cases in which the disease commences under twenty years of age, in certain well-defined signs, or stigmata of degeneration, both anatomical and psychical. On this basis, epilepsy may arise at any age, independently of exciting or determining causes. There are, however, numerous instances in which the disease is ascribed and rightly attributed, to various accidental, or occasional circumstances which will be described in this chapter.

From the etiological standpoint, epilepsy may be divided into two great subdivisions; first, those cases in which the first fit arises spontaneously and is attributed to the normal processes of development and growth, taking place in persons predisposed by heredity to convulsive disorders; and secondly, those cases, fewer in number, which are ascribed to various exciting, or accidental phenomena which precede the first fit and seem to have a causal relation to it.

It is held to be a cardinal principle, that the cause of epilepsy is that circumstance to which the first fit is apparently due. The instability of the brain, once induced, becomes a characteristic feature, so that when epileptic convulsions appear, attacks may, and do, recur quite independently of the original, or any other obvious exciting cause. In this way, the epileptic state, or constitution, becomes established,

a state which is characterised by a tendency to the recurrence of convulsions and their associated mental phenomena.

The determining causes of epilepsy are found in the following:

1. Physiological causes. Of these the most important are puberty, the onset of the catamenia, pregnancy and the puerperium. Sleep would appear to play an important part in the production of epileptic fits, as many instances may be cited in which the disease began and continued for months or years solely during sleep. The ingestion of food also has been found in a few cases to be the only exciting cause of the first and subsequent fits.

2. Psychical causes. The leading psychical causes are found in emotional excitement, fear, shock, anxiety and the effects of overwork, and have an important rôle in the determination of epileptic seizures.

3. Pathological causes. Under this are included the exanthemata and acute infective disorders, organic diseases of the brain and trauma of the head; also morbid conditions of the various organs, such as the eyes, ears, nose, etc., which are associated with the so-called reflex epilepsies. It will be shown that some forms of epileptic manifestation may arise from auto-infection from the alimentary canal, or from disorders of the bodily metabolism.

Any of the above mentioned causes may induce individual attacks, or series of seizures, after the disease has become established. Thus it is not uncommon to find remission from epileptic fits broken by a pregnancy, an acute infective disorder, or an injury to the head; while a temporary increase in the number of the seizures is readily induced during the course of the disease by one or other of the above mentioned causes.

It is, however, necessary to enter with more detail into the frequency of the occurrence and the relative importance of the occasional causes as factors, both in determining the onset of the first fit, and as potent elements in the relapses of the confirmed disease.

In the first place it is possible to demonstrate, by a study of the family and personal history of individual cases, and by an examination into the structural peculiarities, or stigmata of degeneration, that the causes of epilepsy, about to be described, are only potent in individuals of a neuropathic,

or hereditarily acquired, disposition. Without such disposition, it is impossible to conceive how the physiological states of pregnancy and the puerperium, the acute diseases of infancy and childhood, traumatism of the head, emotional shock and fright, and organic affections of the brain, can cause either the acute form of epilepsy in some cases, or the more prolonged and chronic type of the disease in others.

In the study of the occasional causes, and their relation to the several types of epilepsy described in the succeeding pages, attention will therefore, as far as possible, be drawn to the evidence of a neuropathic heredity, either by reference to the family history, or from the evidence of the stigmata of degeneration.

1. The Reproductive Functions.

Menstruation. The reproductive functions in women stand in close relation to the onset and relapse of epileptic seizures. As already shown the most common cause of epilepsy in either sex is the onset of puberty; and in girls the incidence of the disease is usually accompanied by irregularity in the appearance of the catamenia. There is little or no clinical evidence to support the view that the regular establishment of the menstrual functions is favourable to the arrest, or diminution, of epileptic seizures; on the other hand, instances are not uncommon, in which the establishment of the monthly period has been attended by an increase in the frequency and severity of the attacks, and the development of the confirmed malady.

The relation of epileptic fits to menstruation is well-established. In the majority of female epileptics, the seizures are observed to occur immediately before, or shortly after, the monthly period; or if the fit-incidence is frequent, an augmentation in their number, or severity, is noticed at these times. In one patient long periods of amenorrhoea, sometimes physiological, at other times of a spontaneous character, were associated with a remission of the seizures, which invariably returned on the reappearance of the catamenia. The "menstrual type" of epilepsy is clinically important only in young women about the time of puberty.

On the other hand, the menopause is a period in the reproductive history of women, which has a peculiarly small influence upon epileptic attacks. In none of my cases was

an arrest of the seizures directly attributable to the influence of this epoch, although there were a few in which the fits became arrested between the ages of 41 and 45. Nor could it be definitely stated that this epoch is likely to primarily induce attacks. Reference to the age-at-onset table (p. 19) shows that there is no marked increase in the number of cases in which the disease arose between the ages of 40 and 45. As has been said by a writer on epilepsy, women who have passed through the stress and strain of puberty, pregnancy and the puerperium are not likely to suffer from fits at this period, if it presents anything like normal features.

In many cases of essential epilepsy, originating in women at, or after, the climacteric period, careful examination will often reveal a history of "faints," or "giddiness," which would suggest antecedent minor epilepsy, or its equivalent in the form of periodic headache or "bilious attacks."

The influence of *pregnancy*, the *puerperium* and *lactation* has been studied by myself in 41 women with a history of 61 pregnancies; and the results have been tabulated in the following way:

Quickening induced a relapse in	7	cases.
Pregnancy was the original cause in	2	„
„ induced relapse in	14	„
„ was temporarily beneficial in	6	„
„ made no difference in	1	case.
Accouchement was the original cause in	5	cases.
„ induced a relapse in	17	„
Lactation was the original cause in	3	„
„ induced relapse in	6	„
41 *cases.* *Total Pregnancies*	61	

In 16 of the 41 cases, there was no note in the records of any family or other history bearing upon the question of heredity, so that a total of 25 remains, in which this matter was studied. Out of the 25 cases, 13, or 52 per cent., gave a history of family epilepsy or alcoholism—a percentage which is almost parallel to that ascertained, as the relative proportion of a family predisposition amongst epileptics in general (p. 27).

Pregnancy would appear to stand towards the incidence of epileptic attacks in three ways:

(*a*) As the accidental cause of the disease. There were

only two cases of the series in which this occurred, both being young women in their first pregnancies.

(*b*) By inducing relapse after a remission, or by augmenting the frequency or severity of the seizures. Of the series, twenty-one were thus affected, in four of whom, the relapse was clearly related to the time of quickening. Three cases, previously subject only to minor seizures, developed the major type of fit. There were two cases in which the patients had severe attacks in three successive pregnancies, which were followed after confinement by several months of complete freedom from fits. A relapse of fits during pregnancy did not necessarily imply their continuance in a severe form after accouchement.

(*c*) By causing temporary arrest, or amelioration of the seizures. This was observed in six cases. In one instance, although there was freedom from attacks during one pregnancy, the fits became more frequent during a second.

These observations are in general harmony with what is commonly known as to the effects of pregnancy on epileptic seizures. There are undoubted cases on record in which fits have been permanently arrested by pregnancy, and others in which a temporary remission has been observed; but it will be seen from the figures here given, that it is more common to find a relapse of the attacks, or the conversion of a minor type of the disease into the combined major and minor type. Nerrlinger's[1] figures on this subject show that of 92 women with 157 pregnancies, 28 per cent. showed complete cessation of the fits during pregnancy, and 35 per cent. were made distinctly worse.

Accouchement and the *Puerperium* have an important bearing upon the incidence and relapse of epileptic seizures. There were five cases in which the disease clearly originated at this time, and seventeen in which it led to a serious relapse. Of the first series, the onset was in the form of serial epilepsy, or the status epilepticus (puerperal eclampsia), and the disease continued in a chronic form for many years afterwards. In one case it commenced during the fourth confinement; and in three others during the first. These cases are particularly interesting, as they argue strongly in support of the view

[1] Nerrlinger, *Inaug. Diss.* Strasburg, 1889, quoted by Curschman, also Curschman, *Munch. Med. Wochensch.* 1904, nr. 26.

of Féré, that puerperal eclampsia, like many other "eclampsias," is merely epilepsy in an acute form, and that the disease, once started in this way, may persist for years. Two cases were illustrative of this, by the fact that after the original eclamptic attack, the further continuance of the malady was in the form of minor seizures over a period of eighteen and ten years respectively.

Of the second series—those cases in which a relapse was caused by confinement—there were two, in which a remission of twelve and eighteen months respectively, was broken by the eclamptic seizures of the puerperium. In the others, they merely formed an incident in the course of the confirmed disease.

It was not uncommon, in cases of already existing epilepsy, for puerperal convulsions to be delayed until the later pregnancies.

The incidence of serial epilepsy, at or immediately succeeding parturition, is therefore a common feature in epileptic women, and raises the question as to the diagnosis of some forms of puerperal eclampsia. A history of pre-existing attacks would determine the diagnosis of epilepsy; while the existence of a neuropathic family history, or the presence of stigmata of degeneration, would point to eclamptic attacks as being of epileptic nature. The presence of albuminuria does not of necessity form the main element in the differential diagnosis, as albumen has been found in the post-paroxysmal urine of epileptics (Voisin and Péron), although it is not of common occurrence.

It is therefore clear that many cases of puerperal eclampsia are really examples of serial epilepsy, or the status epilepticus, induced during the puerperium in predisposed and neuropathic persons.

Lactation in some cases is conducive to the onset and relapse of epileptic seizures. It was the accidental cause of epilepsy in three cases, and induced relapse in six. In one case, fits ensued during the period of nursing after three successive pregnancies, and ceased on weaning. In two cases the disease which originated while suckling the first baby, returned when nursing the second, and eventually persisted as the confirmed malady. In another instance, suckling a child induced a relapse after a remission of four years.

From these facts it is permissible to state that lactation stands in the same relation to the onset and relapse of

epileptic seizures as pregnancy and the puerperium; and that the disease, if started in this way, may persist as the confirmed malady in those who are predisposed towards it.

2. Sleep.

Sleep is one of the common physiological causes determining the recurrence of epileptic seizures. In many cases fits only occur during sleep, so that a clinical type of the disease has been recognised as *Nocturnal Epilepsy*. Of 177 cases of the present series, in which the fits were carefully observed and recorded, 30, or 16 per cent., had a nocturnal incidence; 50, or 27 per cent., were diurnal cases, and 97, or 54 per cent., were of the combined day and night type. An interesting feature of the nocturnal type lies in the proclivity to convulsion should the patient "have a nap" during the day. In many instances nocturnal epilepsy persists unrecognised for years, until a convulsion in the day establishes the existence of the disease. The nocturnal variety is stated to be more frequent at the commencement of the disease. In the combined nocturnal and diurnal form the attacks occurring during sleep are more commonly of the major, those during the day of the minor type of seizure. (Chart 5.)

A. Pick[1] has drawn attention to the frequency of fits during the hours of deepest sleep, *i.e.* immediately after falling off to sleep, and an hour or two before waking, a circumstance which is stated to be dependent upon the state of the cerebral circulation, as the maximum of brain anaemia occurs during deepest sleep.

My observations upon the relation of sleep to the incidence of epileptic fits are recorded fully in a subsequent chapter (p. 107), but it may be stated here that fits are more common during the sleeping than the waking hours, and that the time of greatest frequency is the hour or two after falling off to sleep. Whether these facts are to be explained on the theory of brain anaemia (Pick), of lessened inhibitory control (Clark), or upon variations in the reaction of the blood (Haig), is a point upon which there is as yet barely sufficient evidence to form an opinion.

Of *causes of epileptic fits*, other than those of a physiological character already described, the following were obtained, with

[1] Pick, *Wiener Med. Wochenschrift*, 1899, nr. 30.

their percentage frequency, out of a total of 388 cases, in which inquiry as to causation was made.

Cause.	Total Cases.	Percentage.	Percentage with Neuropathic Heredity.
Infantile Convulsions	34	8·7	52
Trauma	28	7·2	35·9
Infantile Hemiplegia	23	5·9	—
Acute Infective Disorders	22	5·6	59
Psychical causes	16	4·1	68·7
Miscellaneous	5	1·2	—
No exciting cause known or assigned	260	67·3	—
Total	388	100·0	

3. Epilepsy and Infantile Convulsions.

The most common demonstrable cause of epilepsy is infantile convulsions. Out of 1000 cases of epilepsy, 100, or 10 per cent., commenced during the first year of life; and 34 cases or 8·7 per cent. had convulsions during infancy, and subsequently developed epileptic fits. There is therefore a total of 18·7 per cent. of epileptics, in whom the disease began under the age of twelve months, no doubt, to some extent in the so-called dentition convulsions.

The relation between infantile convulsions and epilepsy has engaged the attention of numerous writers. Féré holds that the eclampsia of children, and epilepsy, are identical, both from the etiological and symptomatological standpoints; and that 34 per cent. of epileptics have had fits in infancy. Gowers believes that the relation is less frequent, viz., 10 per cent.; other writers, without stating figures, refer to the frequency and importance of teething convulsions as antecedents of epileptic fits. In the words of Binswanger, infantile eclampsia prepares the ground for epilepsy.

Infantile convulsions, therefore, occurring in association with the eruption of the first dentition, pyrexia, infective disorders, or gastro-intestinal irritation, should be regarded as danger signals of primary importance, as likely to be preludes of epilepsy in later life. On the other hand, should the convulsions of infancy continue, after the exciting cause has

apparently subsided, or been removed, chronic epilepsy has become established, even though the convulsions be temporarily arrested at three, four or five years of age; for there is no commoner remission throughout the whole course of the disorder than that which takes place during childhood, to relapse at, or about, the onset of puberty.

Infantile convulsions and epilepsy commencing within the first year of life, indicate a strong neuropathic heredity. Other common indications of this predisposition are found in night terrors, laryngismus stridulus, somnambulism and nocturnal incontinence. Of the cases of epilepsy, which commenced within the first twelve months, 22 per cent. had a hereditary history of epilepsy, or insanity; and of the 34 cases with infantile convulsions, 18, or 52 per cent., had a neuropathic heredity.

4. Infective Disorders.

Acute infective diseases were the determining causes in 22 out of 388 cases, or 5·6 per cent.

In all the cases, epilepsy was induced without any demonstrable evidence of organic disease. The diseases which were found to act as exciting causes of the first fit, or as determining a relapse of subsequent seizures, were: Scarlet Fever, Measles, Pertussis, Influenza, Acute Pneumonia, Rheumatic Fever; and less frequently "Septic throat," Abscess, and Herpes Zoster. One patient attributed the onset of the malady to Vaccination. Enteric Fever was given as the cause in one case.

The frequency of Scarlet Fever as a determining factor in the production of epilepsy in young people has been referred to by Gowers and other writers. In this disease, as in the others above mentioned, the first fit ensued during convalescence, or within a brief period of the subsidence of the exanthem, and would seem to be attributable to the influence of toxic substances acting directly upon the cell elements of an inherited neuropathic nervous system; for out of the 22 cases in which the disease was ascribed to these causes, 13, or 59 per cent., had a hereditary history of epilepsy or insanity.

The widespread influence exerted upon the system generally, and upon the nervous structures in particular, by the poisons of Scarlet Fever and of Influenza are well known; and it is scarcely necessary to mention the acute nephritis, endocardial lesions and chorea, which may be associated with the former, or the

persistent headaches and psychoses, which frequently succeed an attack of Influenza.

Measles was the assigned cause in four cases, and Whooping Cough in three. This latter malady, though not in itself a common cause of idiopathic epilepsy, is a not infrequent precursor of infantile cerebral hemiplegia.

Other specific infections, as excitants of epilepsy, are mentioned by various writers.[1] Malaria has been stated by Marandon de Montyel[2] to have induced epilepsy in those who are hereditarily disposed; while Enteric Fever, Diphtheria and Meningitis have been assigned as exciting causes by others.

But there is another aspect of the relation between epilepsy and the acute specific disorders; viz., a tendency to respite which is sometimes temporarily induced during the course of the confirmed disease. There was one case, in which a respite from fits for several months was obtained after an attack of acute pneumonia. Although scarlet fever as a rule increases the number and severity of epileptic seizures, one case was observed in which a mild attack of this disease induced a period of amelioration lasting for several months.

Bourneville[3] records an epidemic of typhoid fever amongst epileptic children, in which the attacks were suspended during the pyrexia, and which persisted for some time after recovery.

As a rule, an attack of acute febrile disorder gives only a temporary respite from fits; no prolonged, or persistent, arrest of the seizures can be predicted.

5. Psychical Causes.

Under this heading are included such accidental circumstances as fright, mental emotion, prolonged anxiety, grief and overwork, all of which are to be found in the records as assignable causes of the first seizure.

The relative importance of the psychical causation of epileptic seizures has been variously stated by different writers. Gowers regards it as the most potent of all the immediate causes of epilepsy; one third of his cases, in which a definite cause was given, ascribed the onset of the disease to fright, excite-

[1] See Féré, *Comptes Rendus*, 1893. Lannois, *Neurol. Centralblatt*, 1894, p. 871. P. Marie, *Semaine Médicale*, 1892, No. 36. Voison, *Semaine Médicale*, 1901, No. 12.

[2] Marandon de Montyel, *Rev. de Médicin*, Dec. 1899.

[3] *Progres Médical*, Sep. 1899.

ment, or anxiety. Russell Reynolds also states that the conditions acting psychically are much more commonly found occupying a causative relation, than are any other. Spratling traced a psychical cause in only 5·5 per cent. of his cases; while Féré, Binswanger and others refer to the importance of emotional circumstances as exciting to epileptic fits in predisposed persons.

In my series of 388 cases, psychical influences were assigned as the cause of epilepsy in 16 cases, or 4·1 per cent.; of whom 11 were females, and only 5 were males. That this cause acts upon an underlying neuropathic basis is seen from the fact that 11 out of the 16, or 68·7 per cent., gave a family history of hereditary influences.

The different forms of emotional influence are seen to act at different ages: thus we find that of the four cases in which "fright" was stated to be the cause, all were girls under twelve years of age, and the first fit occurred within a few hours.

Of those attributed to "shock," which referred more especially to the news of the sudden death of a near relative, all were adults over twenty-one years of age; while the same factors were noted in those who assigned their seizures to overwork and worry. In the latter category the patients were, with two exceptions, over forty years of age.

The sight of another person in an epileptic fit, has been stated by some writers to have been the cause of the first fit in others. I have not encountered this either as a cause, or as inducing a relapse. In my experience, epileptics are ever ready to assist one of their fellows, who has been taken in a convulsion. On the other hand, in hospitals where epileptics and hysterical patients are warded together, an epileptic convulsion may give rise to quite an outburst of hysterical attacks in those who are subject to them.

6. Trauma—"Traumatic Epilepsy."

Injury to the head caused by a fall or a blow, is a commonly assigned cause for the onset of epileptic seizures. In not a few cases the onset of epileptic fits, at or about puberty, will recall to mind a fall on the head long forgotten, but to the influence of which the subsequent seizures will be ascribed.

There will also be found epileptics, in whom the head has received no direct injury during a fall from a small height

on to the ground, ascribing the onset of their disease to a general shaking which the nervous system has in this way received.

In these two types of cases there exists little, if any, relation between the assigned cause and the malady.

On the other hand, a number of cases of genuine idiopathic epilepsy are correctly attributed to a blow, a fall, or other severe injury to the head. In these cases we have to distinguish between a shaking, or concussion, of the brain without coarse lesion, and a definite organic injury to the skull, the membranes, or the brain itself. The influence of general trauma as a factor in the causation of well-marked and prolonged nervous disorders is well known, as is seen in the production of the traumatic neuroses and traumatic hysteria. The action of trauma, also, upon chronic nervous disease is well established, as may be seen by the effects of a fall or blow upon the head in tabes dorsalis, general paralysis, or in already existing epilepsy.

The influence of injury of the head in the causation of epilepsy has therefore to be studied in two sets of cases.

1. Those without an obvious organic lesion of the brain.

2. Those with an organic lesion of the brain, skull, or membranes.

1. "Traumatic epilepsy" without obvious organic lesion. Injury to the head in these cases may be supposed to act in much the same way as emotional shock or fright. Its influence, however, can only be potent for a limited period, so that cases in which a history of trauma is given, should only be described as such, provided the injury preceded the onset of the first fit, by a few days, or weeks, or at the most a month or two.

The period intervening between the infliction of the injury and the first fit, is so vitally important in the relation between cause and effect, that the following table has been constructed to show the duration of the interval in the 19 cases, in which trauma was the assigned cause:—

Interval.	Number of Cases.
At once	5
Within a week or two	8
Within two or three months	3
About 1 year	2
About 7 years	1

The majority of cases of traumatic epilepsy without organic

lesion, therefore, arise within a few (2 or 3) months after the receipt of the blow. In how far epilepsy arising a year or more after head trauma should be described as "traumatic epilepsy," is a point upon which difference of opinion may exist. Binswanger has stated that between the injury and the onset of epilepsy many years may intervene, but it is safe to say that if more than a year elapses between the receipt of the injury and the first fit, an organic lesion is probably the cause of the fits.

In all such cases, the question of heredity and a neuropathic disposition, and its influence as the main causative factor has to be carefully weighed.

In my series of 388 cases, trauma of the head was the assigned and only demonstrable cause of subsequent epilepsy in 28, or 7·2 per cent. Of these, 19, or 4·2 per cent., presented no evidence of any local injury to the bones of the skull or the brain;[1] while in the remaining nine, an organic lesion of the skull, or the brain, or both, was present. Of the nineteen cases, without organic lesion, six gave an hereditary history of epilepsy or insanity, while of the cases with gross lesion four gave a neuropathic history, as shown by table 17. This gives a percentage of 35·9 of the traumatic cases with a neuropathic hereditary history.

There is a type of epilepsy following head injury, in which no organic lesion of the brain or skull may be apparent, although examination of the head long after the infliction of the injury may reveal a tender spot, or a scar, and in which the fits may be either of a general character, or present "aura symptoms" of the Jacksonian type. Subsequent examination of the brains in these cases, either in consequence of operation or on post-mortem, sometimes shows the existence of an ill-defined cystic tumour, a sclerosed patch, or an adhesion between the membranes and the cerebral cortex, changes which are no doubt the direct consequence, hemorrhagic or inflammatory, of the original injury. The interval which elapses in such cases, between the trauma and the first fit may be prolonged. This is an important type of "traumatic" epilepsy, and will receive further consideration under surgical treatment (p. 256).

[1] Of 210 cases of epilepsy collected by Wildermuth, 3·8 per cent. were ascribed to cranial trauma, where there was no local evidence of injury, and Finckh (*Arch. f. Psychiatrie*, 1905, p. 820) found 17·6 per cent. of traumatic cases.

2. Cases with traumatic lesions of the skull or brain.

Table 17 *showing the lesion, age at onset, hereditary history, and interval between the infliction of the injury and the onset of Epilepsy.*

No.	Lesion.	Interval.	Age at Onset.	Heredity.
1	Fracture of skull - -	5 days	18	Father epileptic
2	do. -	1 week	6 months	Sister defective
3	Probable fracture - -	3 weeks	—	None known
4	Fracture of skull and hemiplegia - - -	7 weeks	34	None known
5	Gunshot wound - -	2 months	28	No note
6	Fracture of skull - -	3 months	8	None known
7	Gunshot, of head - -	5 months	27	Sister insane
8	do. - -	1 year	27	Brother epileptic
9	Fracture of base with slight hemiplegia -	6 years	13	No note

Of these there were nine cases. Five gave a history of fracture of the skull with or without paralysis. Three were cases of gunshot wound of the head, without obvious motor paralysis, but one of them was a gunshot injury of the occipital region, in which there was a quadrantic hemianopsia, as the only paralytic phenomenon. One was a case of severe gunshot wound of the skull, with splintering and depression of the bone and consequent hemiplegia.

The interval between the receipt of the trauma and the onset of the seizures was variable, as is seen from the above table, where the nature of the lesion, the age at onset of the fits and the hereditary history is also stated.

Of the nine cases, four gave a hereditary history of epilepsy, insanity and mental defect; in three there was no known history, and in two there was no recorded note.

The age at onset of the seizures in the present series is somewhat later than that usually found, owing no doubt to the circumstances under which the injuries were inflicted, four of the patients having received gunshot wounds of the head during the late South African War.[1]

The interval between the traumatism and the onset of the

[1] I am indebted to Dr. Ferrier for the notes of three of these cases. Allen (*Boston Med. and Surg. Journal*, 1906, p. 396) states that of 167 cases of skull injury received in the American Civil War, 23, or 13·7 per cent., were known to suffer from subsequent epilepsy, and in the Franco-Prussian War, of 571 cases, 25, or 4·3 per cent., were similarly affected.

seizures in this series of cases presented the same variations as in the cases without organic lesion, in eight out of the nine cases the seizures commencing within a year of the receipt of the head injury.

The type of seizure may be either the major or minor variety: in one case there was a combination of both grand and petit mal. In cases with hemiplegic symptoms, the convulsion usually affects the paralysed more than the non-paralysed limbs, and may be preceded by a local aura, more especially in the paralysed arm. Sometimes the seizures are of great severity, and in two of the cases, there was marked mental deterioration. In one case the attacks seemed to be of a psychical character.

7. Syphilis.—"Syphilitic Epilepsy."

Syphilis is stated to be a causal factor in the production of epilepsy in three ways:

1. Congenital Syphilis.
2. Secondary Syphilis.
3. Organic syphilitic lesions.

1. *Congenital syphilis.* Opinions differ as to the importance and frequency of congenital syphilis in the causation of idiopathic epilepsy. Kowalevsky has stated that it is a frequent cause, the syphilitic poison sometimes producing chemical molecular changes in the nerve cells favouring the development of convulsions, at other times leading to defect and backwardness in the development of the brain. Hence, according to the extent and intensity of the action of the syphilitic virus, diverse clinical appearances are developed in those who are the subjects of the inherited disease. On the one side, there may be all degrees of mental impairment from idiocy and imbecility to the slighter forms of mental defect and neurasthenia; and on the other, intermediate types of nervous disorder are seen in epilepsy, chorea, and hysteria.

Gowers, on the other hand, states that he is unable to perceive definite grounds for suspecting that epilepsy is a consequence of inherited syphilis, except as an indirect effect of its influence on general development. Any influence of syphilis, exerted through a "toxic blood state" upon the nervous system, would favour this result; but in very few instances did he find the

attacks to begin in infancy: in most cases they commenced towards the end of childhood, or later.

Although the frequency of imbecility and idiocy as a result of syphilis in a parent is admitted by most authors, yet, according to many, the signs or stigmata of congenital syphilis in imbeciles are seldom well developed.

But in order to establish the direct connection between inherited lues, idiopathic epilepsy, and congenital mental deficiency, definite and characteristic clinical signs of the taint ought to be apparent.

Out of the series of 1000 cases of idiopathic epilepsy, collected from various sources, both hospital and private, I was able to find only four cases presenting the typical signs of congenital lues,—the Hutchinsonian teeth and the characteristic face. Of these, one was a male and three were females. The fits commenced at the usual time, from twelve to nineteen years of age; and two out of the four had an hereditary history of epilepsy. The fits were of the usual character, in three cases major seizures, in the fourth the combined major and minor type. Two of them responded well to treatment by the bromides, while a third became a confirmed epileptic. Two showed considerable backwardness from birth and mental deterioration later, while the third and fourth retained their mental activities.

In none of the cases of infantile hemiplegia with subsequent epilepsy were there any clinical indications of hereditary syphilis; the rôle, therefore, of this disease in the production of the lesions underlying infantile hemiplegia would appear to be entirely subsidiary, if indeed it exists at all.

I am therefore led to the conclusion that *inherited syphilis plays little part in the genesis of idiopathic epilepsy.* As already shown, the cases in which there was clear clinical evidence of syphilis, epilepsy did not commence earlier than under other circumstances; the tendency towards dementia and the confirmation of the malady was not more striking than in ordinary epilepsy, and the presence of hereditary lues did not necessarily interfere with successful bromide medication.

2. *Secondary syphilis* has been stated to exert an exciting influence in the causation of epilepsy in some cases. According to Fournier genuine epileptic seizures may occur in the so-called

"secondary" stage, in which there is neither clinical, nor anatomical, evidence of the presence of any organic syphilitic affection of the brain or its membranes. In these cases epileptic seizures may be ascribed to a syphilitic "dyscrasia," or intoxication of the nerve centres by the syphilitic poison, and may be comparable to the instances of syphilitic insanity, which have been described as occurring during the eruptive stage of the disease. Evidence upon these points is extremely meagre, but it ought not to be forgotten that in the "blood stage," the syphilitic poison may act upon the neurones in the same way as the toxic element of scarlet fever, measles, and the other exanthemata, in those who have a neuropathic predisposition to epilepsy.

In my series of cases, there was only one patient, a male, *act.* 28, in whom the disease commenced during the "secondary" stage, four months after the appearance of the primary sore; and evidence of a neuropathic heredity was supplied by the fact that the patient's brother suffered from melancholia.

If syphilitic infection may be an exciting cause of epilepsy, similar to the other exanthemata, so also may it aggravate the frequency, or intensity of epileptic fits in those subject to the disease.

3. *Epilepsy consequent upon local organic syphilitic lesions of the brain, or its membranes.* This is one of the forms of "organic epilepsy," in which convulsions, having characters similar to those of the idiopathic disease, are associated with, and are probably dependent upon, an organic lesion either of the brain, its membranes, or its blood-vessels.

Any organic lesion of the brain may theoretically give rise to convulsive seizures, which may be followed by recurring attacks of an epileptic character. This, however, is not usual, except in cases of infantile hemiplegia, and in the vascular and gummatous lesions associated with cerebral syphilis.

Reference is not made in this connection to the well-known epileptiform, or localised convulsions, associated with the name of Hughlings Jackson, but to generalised convulsions of an epileptic character, following in the wake of organic lesions.

Two cases illustrating this association may be recorded:

(1) A young man, *act.* 30, seven years after syphilitic infection was suddenly seized with an epileptiform attack and loss of consciousness, which was followed four months later by another

of a similar character. One month after the second fit, a partial convulsive seizure occurred involving the left arm and side of the face, and was followed after some hours by a sudden attack of unconsciousness and indistinct speech. During the following year, although under treatment, he had several partial seizures, sometimes with, at other times without, impairment of consciousness. During the next twelve months he had on many occasions ordinary epileptic attacks, and these have continued to recur two and a half years after the original seizure. There was no epilepsy known in the family, nor any previous personal history of fits.

(2) A man, *aet.* 38, nineteen years after syphilitic infection was suddenly seized with left-sided hemiplegia, followed by stupor; he recovered from this in a month. A year later he had a right-sided hemiplegia with aphasia, from which he partly recovered. Six months after the second attack he had an epileptic fit, and these have recurred from time to time for three years after the original stroke.

These two cases are typical instances of ordinary epileptic seizures, following upon destructive cerebral lesions of a syphilitic character, the former probably due to a gummatous new growth, and the latter to a vascular occlusion.

The absence of any neuropathic family history in these cases does not argue in favour of its non-existence, nor does the presence of organic lesions exclude the influence of a neuropathic predisposition in the causation of the symptoms.. Féré, indeed, states that a large number of syphilitics suffering from epilepsy belong to neuropathic families. Just as genuine idiopathic epilepsy arising in later life, in those who are predisposed by heredity, may be due to an infective disorder, or a mental or physical strain, so in the syphilitic the latent predisposition may remain in abeyance until an organic cerebral lesion evokes the convulsive tendency.

Hence the general epileptic convulsions of "organic" epilepsy, whether arising from local syphilitic disease of the brain, or its blood-vessels; from trauma of the skull or brain, as already described; or from the destructive changes, which give rise in early life to infantile hemiplegia, possess not only the clinical features of those found in the idiopathic disorder, but arise in many instances in those who are subjects of a neuro- or psychopathic disposition.

8. Infantile Hemiplegia.

Closely allied to infantile convulsions as a determining cause of subsequent epilepsy, are the "cerebral birth palsies," more especially infantile hemiplegia. This was the cause of the malady in 23 out of 388 cases, or 5·9 per cent. Of these cases, there were four in which the paralysis was stated to have dated from birth, or during the first few weeks of life. In ten, hemiplegia developed between six and twelve months of age; in six, between the first and second birthdays; and in three, between the second and third.

The onset of hemiplegia is ushered in by convulsions, usually of a severe character, which cease for a time, occasionally for several years, but in the majority of cases they return in the form of epileptic seizures, either of the major or minor type, or of the combined variety. In the present series of cases the average age for the onset of the subsequent epileptic attacks was from six to eight years, the longest interval between the hemiplegia and epilepsy being twenty-one years.

This is a form of epilepsy in which the original cause of the malady is a local cerebral destructive lesion. The nature of the lesion varies; in those in whom the hemiplegia dates from birth, or shortly after, the brain may have been damaged during parturition from meningeal hemorrhage; in others, occurring at a later period, thrombosis of the cerebral blood-vessels, leading to softening and cystic formation of the cortex and subcortical tissue, has probably occurred. Although rarely observed pathologically at the onset, the subsequent effects of thrombosis in these cases are seen in porencephaly, a not uncommon pathological lesion in those who have suffered from infantile hemiplegia and epilepsy. In others, again, the lesion has been stated to be an acute encephalitis, comparable perhaps to what is seen in the acute anterior poliomyelitis of children, while embolism is assigned as the cause by other investigators.

The evidence of heredity in these cases is not always clear. I found it present in only three cases, and in four no hereditary neuropathic history was known; its existence was probable in several others from the presence of well marked stigmata of degeneration.

The seizures which subsequently develop in these cases are

epileptic in character, the convulsion having a tendency to implicate the paralysed limbs, in excess of the non-paralysed. There is frequently a local aura, beginning in the paralysed hand or arm. Attacks of minor epilepsy, or "petit mal," are not infrequently seen. In one instance they existed for some years before the convulsive seizures developed.

The mental condition is usually that of pronounced dementia. The patients are backward in walking and talking; they are often passionate, mischievous, and destructive.

The percentage of infantile hemiplegics that become epileptic is variously stated by writers on the subject. In my series epilepsy developed in 78 per cent. of the cases of Infantile Cerebral Hemiplegia.

9. Organic Epilepsy.

This is a term applied to cases characterised by recurring epileptic seizures, associated with organic lesions of the brain, and continuing after the active stage of the disease is over. The seizures present the characters common to those of idiopathic epilepsy, having the features of the major or minor fits, or psychical attacks. Associated with these recurring convulsions, there is usually some degree of hemiplegia, more or less pronounced. If paralysis is present, the convulsion may start in, be limited to, or involve more particularly, the paralysed limbs, but in whatever way convulsion originates, consciousness is early and completely abolished. Sometimes the malady is characterised only by recurring major convulsions, at other times the minor type of seizure is alone present, while in other cases there is a combination of the major and minor types. The convulsions, once established, may persist throughout the remainder of life as confirmed epilepsy.

Epilepsy is met with in the following varieties of organic cerebral disease:

(1) Cases of epilepsy due to, and arising from, traumatic lesions of the skull and brain.

(2) Cases of epilepsy sequential to focal organic cerebral lesions, such as are induced more especially by acquired syphilis.

(3) Cases of epilepsy associated with, and due to the cerebral destructive lesion causing infantile hemiplegia.

(4) Convulsions having the general characters of idiopathic epilepsy, and present either as an early symptom of intracranial

tumour, or persisting as confirmed epilepsy after the original exciting cause has been removed.

10. Miscellaneous Causes.

Many causes are assigned as factors in the production of epilepsy and of recurring epileptic fits, which have in all likelihood little or nothing to do with the onset of the disease. There are, however, others, not included amongst the preceding, which act as occasional determining agents and require brief consideration; but it should be borne in mind that these are only efficacious in the production of fits, when a hereditarily acquired disposition to the disease already exists.

(*a*) *Toxaemic* influences arising in the gastro-intestinal tract, in connection with the processes of digestion, have within recent years received considerable attention as causes of epileptic fits. There is some evidence, which will be referred to on a later page, (199), to show that a type of epilepsy may be ascribed to such influences. There is, however, little known as to their nature or their action, and treatment directed more especially towards intestinal asepsis has not produced modification or cure.

(*b*) *Reflex causes.* A variety of the disease has been described as *reflex epilepsy*, in which a lesion of a peripheral nerve has led to the onset of the first epileptic attack, and consequent development of recurring epileptic seizures. It is more common to find cases of already existing epilepsy in which peripheral irritation of various kinds may bring about the recurrence of seizures, or in which a source of local irritation may primarily start convulsions in a person hereditarily and structurally predisposed to epilepsy.

It is to the latter type of case that attention is directed, as it seems to form a not uncommon cause of some of the epilepsies found in children and young adults. It is in cases, also, with an initial warning, referred, for example, to an organ of special sense, that a local peripheral lesion may be discovered, removal of which materially diminishes, or even arrests, the epileptic seizures. The arrest may only be temporary, as convulsions, once induced by whatever cause, may persist as confirmed epilepsy, even after the removal of the original exciting irritation.

1. *Nose.* The nares are frequently the seat of adenoid

growths, polypi, or foreign bodies, removal of which should always be effected, even though an aura referred to the olfactory nerve, or nasal mucous membrane, is not present.

2. *Eyes.* Errors of refraction, particularly astigmatism, are common in epileptics, and if high, form one of the not infrequent stigmata of degeneration. Their correction in many instances has been found to be highly beneficial.[1]

3. *Ears.* External or middle ear disease is occasionally co-existent with epileptic seizures. In one case the onset of the seizures immediately followed unilateral acute otitis media; in another, an aura of a "confused sound of voices" was associated with old middle ear disease and deafness upon one side. The relation between labyrinthine disease and epilepsy is referred to later (p. 209).

4. *Teeth.* Epileptics are notoriously prone to carious states of the teeth, which should have careful attention; but I have never seen a case of epilepsy in which the fits have been arrested by removal of those that were carious.

5. *Stomach and intestines.* The digestion of food is, in some cases of epilepsy, a cause of recurring convulsions. There were several cases in which the fits only ensued after a meal, more especially the midday meal, when meat was eaten. In another case, a woman with a strong neuropathic heredity, the cause of the first fit was ascribed to an indigestible meal eaten late at night, but attacks continued to recur without similar, or any, irritation.

In the cases ascribed to intestinal worms, the fits frequently persisted after the worms had been satisfactorily removed.

6. *Genital organs.* The presence of a tight prepuce, with the irritation produced by the retention of the secretions, is a well-recognised cause of fits in boys. Its removal is frequently followed by great improvement, if not indeed by complete arrest. Circumcision should be done in all such cases.

Masturbation is an ascribed cause of epilepsy, but it is doubtful whether it is ever the exciting cause of convulsions. As an associated symptom in epilepsy, as in all forms of degeneracy and mental defect, it is of common occurrence. As a habit it is frequently continued long after the disease has become

[1] Work Dodd (*Brain*, 1893, p. 534) found that out of 52 cases of epilepsy, after correcting the errors of refraction by suitable glasses, 36 showed marked improvement, while 13 showed an arrest of the fits for a time.

confirmed. In some cases it is due to the irritation of a long, or a tight, prepuce.

(*c*) *Tobacco intoxication* as an exciting cause of epilepsy is doubtful; but I have in several cases found excessive smoking, especially of cheap cigarettes, an excitant of individual attacks. In one epileptic, more particularly, small series of fits were induced in this way, series which subsided permanently, when he was no longer in a position to acquire tobacco. Spratling refers to its influence in cases about the time of puberty, and others have recorded instances in which it appeared to have a causal influence in the development of the disease.[1]

(*d*) *Alcoholic intoxication* induces, more especially in predisposed persons, a form of eclampsia, or acute epilepsy, chiefly in conjunction with delirium tremens. Epilepsy is associated with delirium tremens to the extent of about 33 per cent., according to the statements of Westphal, Fürstner, and Moeli. This form of acute epilepsy, due apparently to the direct toxic influence of alcohol upon the cerebral tissues, is rarely followed by epilepsy of the usual chronic form, the convulsions passing away under treatment, when alcohol is no longer permitted, but returning with each attack of alcoholic delirium.

Ordinary chronic alcoholism is not commonly a cause of epileptic fits; on the other hand, in France, where absinthe drinking is prevalent, "absinthe-epilepsy" is stated to be of frequent occurrence; and as already noted on a previous page, according to the French writers, parental alcoholism is the most common cause of epilepsy in the offspring.

The relatively potent influence of alcohol upon the brains of epileptics should not be overlooked in this connection. Alcoholic intoxication in these persons produces serious and severe relapse of epileptic fits, which under suitable treatment may have undergone satisfactory remission, and only recur when the patient is again brought under the influence of alcohol.

There were only two cases in the series, which could be described as examples of alcoholic epilepsy, or epileptic convulsions occurring in association with alcoholic bouts, although there was no attack of delirium tremens. They were both males of adult age, who had not suffered from epilepsy in childhood, but in whom convulsions of a generalised epileptic character ensued as a result of acute alcoholic intoxication.

[1] Bychowski, *Neurol. Centralbl.*, 1900, p. 933.

General Summary.

An endeavour has been made in the preceding pages to show, that the epileptic tendency is a sign or stigma of a neuropathic inherited disposition, the anatomical basis of which is seen in certain well-defined structural peculiarities both of the body and of the cerebral cortex.

In those who have inherited the epileptic tendency, a "convulsive habit" may be established either in the course of natural development, or as a result of certain occasional, or accidental, causes. Once the convulsive habit has been established there is a tendency to its perpetuation in the form of recurring epileptic seizures. Thus, infantile convulsions are frequently the starting point of subsequent epilepsy, either as a direct sequence of the convulsions, or in later years at or about the onset of puberty.

The eclampsia associated with the onset of infantile hemiplegia, was in 78 per cent. of the cases followed by epilepsy in later life.

The convulsions associated with the exanthemata, acute infective disorders, and disorders due to toxic influences, including some forms of puerperal eclampsia, are to be regarded as manifestations of acute epilepsy.

The convulsions arising as a result of trauma of the head, without organic lesion of the brain, and from psychical causes, are of a similar character, but are less often accompanied by an acute onset.

The convulsions induced by traumatic lesions of the brain, by coarse lesions of a vascular, more especially syphilitic, nature, and those of organic cerebro-cortical disease, particularly tumour, may be the forerunners of genuine epileptic seizures, which persist even after the exciting cause has been medicinally or surgically removed.

The convulsions of later life, as are seen in the so-called "Senile Epilepsy," and in those associated with cerebral thrombosis or hemorrhage, point to the existence of a latent convulsive tendency, which is only brought into prominence by an accidental circumstance, or constitutional causes.

The percentage frequency of the hereditary factor, in those cases in which the onset of fits is attributed to an occasional determining cause, is seen to vary from 35·9 per cent. in the traumatic cases to 68·7 per cent. in those of psychical origin.

CHAPTER IV.

THE CLINICAL STUDY OF EPILEPTIC FITS.

Classification of epileptic fits—Degrees of convulsion—Prodromata—Fits with peripheral warning; with visceral warning; with head sensations; with psychical warning; with warnings of special sensation; with sudden loss of consciousness—The complete epileptic fit—Immediate sequelæ.

Classification.

Epileptic attacks reveal themselves clinically in a variety of ways, and authors have adopted various methods of descriptive classification. The old division into "petit mal" and "grand mal," although simple, and in many ways satisfactory, is not found to be sufficiently detailed, for there exist degrees of both the grand and the petit mal.

In Binswanger's work[1] upon Epilepsy, we find the following classification of the clinical manifestations of the disease:

(1) The completely developed fit, *epilepsia gravior*, consisting of:

- (*a*) The typical classical seizure, and
- (*b*) The atypical attack.

(2) The rudimentary fit.

(3) Abortive attacks.

(4) Psychical epileptic equivalents.

Féré,[2] on the other hand, divides the paroxysms primarily into those which are partial and those which are generalised, the latter being further subdivided into:

- (*a*) The complete attack.
- (*b*) The incomplete attack.
- (*c*) Abnormal attacks.
- (*d*) Isolated symptoms.

[1] Binswanger, *Die Epilepsie*, Vienna, 1899.

[2] Féré, *Les Epilepsies*, Paris, 1890.

Much, however, depends upon what is held to constitute an epileptic fit. It is therefore of primary importance to decide what feature, or features, are essential to the recognition of epilepsy.

(1) A sudden fall is not essential. If a person falls suddenly with loss of consciousness, there is usually little doubt as to the nature of the malady, but many sufferers from minor epilepsy never fall; and in some cases of aural vertigo,[1] there is a fall, with loss of consciousness.

(2) Muscular spasm is not a *sine qua non* of the disease. There are psychical attacks of typical epileptic character, in which tonic or clonic spasm, even to a limited extent, is entirely absent; and in the incomplete forms of minor seizure (aura) no spasm is necessarily present.

(3) *The feature necessary to establish the existence of epilepsy is sudden, temporary loss, or impairment, of consciousness.* But yet there are single epileptic phenomena, which occur without loss of consciousness. Thus it is well known that some forms of the minor seizure, in which only the aura is present, may be unaccompanied by loss of consciousness; but in such cases evidence of other attacks will be found in which consciousness is obviously lost. There is also a type of attack, characterised by sudden muscular jerks, or jumps, so violent as to cause a fall, or to make the victim suddenly drop what is in his hands, which may be unassociated with loss of consciousness; but these rarely occur alone, being usually pre- or inter-paroxysmal epileptic symptoms.

It does not therefore appear to be legitimate to diagnose epilepsy, unless there is evidence of loss, or impairment of consciousness; although, as will be shown in the sequel, inter-paroxysmal phenomena may be found in epileptics, in which consciousness is not obscured.

In the *complete* epileptic fit there is loss of consciousness with muscular spasm and convulsion of a tonic and clonic nature, followed by a characteristic after-stage of stupor. These attacks may, or may not, be preceded by an aura or warning. There are many fits, on the other hand, in which either the tonic, or the clonic, stage may be slight, and which should be regarded as *incomplete major* seizures. There are also attacks

[1] Reference to be made to chapter on "Differential Diagnosis: Epilepsy and Aural Vertigo," p. 209.

in which the whole seizure only consists of the "aura" without loss of consciousness—*incomplete minor* fit; and there are other attacks in which the aura is followed by some degree of impairment of consciousness, without obvious convulsion, but sometimes accompanied by a fall. These are *complete minor* attacks. The names given to some minor seizures, by those who suffer from them, vary with the character of the attack: "faints," if the attack is associated with a temporary blurring of consciousness; "sensations," if it is mainly of the nature of a warning; "turns," should giddiness be the most prominent sensation.

There is a third variety of epileptic seizure, in which the spasmodic element is slight and transitory, or is entirely absent, but in which the outstanding feature is revealed in automatic or semipurposive movements, consciousness being obliterated. This is *psychical epilepsy*, which will receive consideration later on under the mental, or psychical equivalents of epileptic attacks (p. 130).

The following is therefore the classification of epileptic attacks, adopted in this work:

(1) Minor epilepsy, { Incomplete attacks. / Complete attacks.
(2) Major epilepsy, { Incomplete attacks. / Complete attacks.
(3) Psychical epilepsy.

Epileptic attacks.

Herpin has been largely followed in the subjoined clinical descriptions of epileptic seizures, as met with in practice. In his book *des Accès Incomplets d'Epilepsie*,[1] an attempt was made to show the close relation and sequence, which exist between the several manifestations of the epileptic paroxysm; and two axioms were laid down by him, the full recognition and appreciation of which have done much to permit of a clear conception of the many and diverse types, which form the attacks of this extraordinary malady. His work was published some years before the epoch-making investigations of Hughlings Jackson upon convulsive seizures, by which the seat of convulsions was clearly located in disorders of the cerebral cortex. Herpin conclusively showed, first, that the incomplete attacks, *e.g.* the

[1] Herpin, *des Accès Incomplets d'Epilepsie*, Paris, 1867.

cramps, partial convulsions, spasms, giddiness, etc., which occur irregularly in the intervals between the major attacks in epileptics, are the complete seizures reduced to their initial symptoms; and, secondly, the incomplete attacks, however diversified they may be, are always or nearly always, similar in the same subject.

Many subsequent clinical observations have amply established the law of identity, thus formulated by Herpin, that the incomplete attacks and the initial symptoms of the complete seizures are identical in the same person, and represent consecutive, though aborted, phases of the epileptic paroxysm.

But before passing to a more detailed consideration of the several manifestations of the epileptic fit, it may be stated in general terms, that epileptic attacks are primarily subdivided into three phases, or degrees, of seizure.

1. The aura.
2. The incomplete fit.
3. The complete fit.

1. The *aura* (*prélude* of the French writers) is the initial symptom of the attack, whether this be complete or incomplete. Thus it may constitute the whole seizure, in which event it forms the incomplete variety of the minor fit. For example, in cases where the major fit is ushered in by a cramp-like feeling, or sensation of movement, in the hand, the minor fit may be represented merely by a sensation of cramp, or of movement, in the hand, with, or without, obscuration of consciousness. Or the fit may consist of a sensation rising from the epigastric region towards the throat and head, at which point consciousness is abolished with the advent of the convulsive movements. In such instances the minor fit consists of the epigastric sensation only (aura), or of its extension upwards as far as the throat and head with loss of consciousness (complete minor).

2. *The incomplete attack* (minor fit; *accès* or *vertige* of the French writers) consists of the aura or warning, with a further development towards the phenomena of the complete seizure—a phase of paroxysm, which may, or may not, be associated with loss, or obscuration of consciousness. Thus an epileptic suffered from convulsive attacks, in which the aura consisted of an abnormal sensation of flavour; but he also had

attacks in which the olfactory aura was succeeded by a feeling of uneasiness in the stomach, and a "dreamy" state (complete minor). Another had fits in which the sequence, or march, of the phenomena was: a sensation rising from the stomach, a sense of choking in the throat, arrest of respiration, loss of consciousness, and tonic followed by clonic convulsions of the muscles of the left arm and leg (major fit). In this case there were also attacks in which the fit ended at the stage of arrest of respiration, in a dazed mental condition (minor fit). A common form of incomplete attack is seen in fits, the aura of which is a feeling of giddiness followed by a sudden fall from loss of consciousness, but without convulsion. In these cases there are commonly found attacks of giddiness (aura), as well as complete seizures, in which the fall is followed by the usual convulsive phenomena of a major fit.

An epileptic, whose major fits were heralded by an aura of a sensation of drawing up of the left leg, had minor attacks of a like sensation associated with a feeling of faintness; another, whose complete attacks presented a warning of stabbing pains in the belly, had incomplete attacks characterised by stabbing pains with a dazed sensation in the head; and a third, in whom the complete seizures were preceded by "a terrible feeling of a disagreeable character," had some attacks consisting only of "the terrible feeling," (aura) and others of this feeling with loss of consciousness but without convulsion (complete minor).

3. *Complete attack* (major fit; *crise* of the French writers). This forms the classical epileptic fit, in which the victim falls from sudden loss of consciousness, and his body is invaded by muscular convulsion. These attacks may, or may not, be ushered in by the initial phenomena of an aura. (For a fuller account of the complete attack, see page 84.)

In many persons, major epileptic attacks are of such sudden onset that the patient is unaware of their occurrence, except from the accidental biting of the tongue, or injury caused by the sudden fall, or from finding himself lying in some unusual position. In these attacks complete abolition of consciousness is the earliest symptom, to be followed almost at once by the stages of tonic spasm and clonic convulsion.

Instances might be multiplied to show that the primary types of epileptic attacks denote consecutive stages of the same series of phenomena—stages which may be arrested at any period, and

demonstrate the truth of Herpin's law that, although attacks vary infinitely in different persons, they are always, or nearly always, similar in the same patient; and that the incomplete attacks and the initial symptoms of the complete attacks are identical.

The warning spreads almost always, though not invariably, from the periphery of the body towards the head. The sensation ascends from the hands or the feet, from the thoracic, epigastric, or abdominal regions, or from the organs of special sense. In many cases it is stated that when the sensation reaches the head consciousness is obscured or abolished. There may, however, be variations of this; sometimes consciousness is abolished at an early stage of the fit, at other times general convulsion of the voluntary muscles commences before consciousness is lost, while in other cases, and they are the more common, loss of consciousness occurs just before, or synchronously with, the onset of muscular spasm or convulsion.

In attacks with initial warning, some peripheral (reflex) irritation may be found to act as an exciting cause of the seizures. Too much weight ought not to be put upon this, but in some instances it presents a possible explanation of the phenomena. Thus in cases with olfactory aura, an examination of the nose will sometimes reveal the presence of polypi or adenoid growths, removal of which may materially assist the other methods of treatment; in those with visual aura, errors of refraction should be systematically looked for and corrected; in cases with auditory warning, deafness from middle, or internal, ear disease will occasionally be found. The close relation which exists in some cases between the ingestion of food and the onset of attacks, points to possible sources of irritation in the gastro-intestinal tract.

Prodromata.

Major epileptic fits, or series of fits, are in many cases ushered in by certain phenomena of a psychical, motor, sensory or somatic character. The proportion of cases in which these prodromal, or precursory, symptoms are present, varies considerably, but it has been estimated that they are found in from 10 to 15 per cent. of all cases of the major type of the disease.

The prodromal symptoms are in many cases solely of an objective character, their existence being recognised by those

who are in constant attendance upon epileptics, and interpreted as certain signs of the coming fit. In only a few instances are they solely subjective, so that the patient is himself alone aware of their occurrence. Of the latter, perhaps the most frequent are headache and a tendency to restlessness at night, and bad dreams.

1. Under the *psychical* prodromata, to which further reference will be made on a later page, are to be found most of the indications of the approaching storm. These are largely of the nature of alterations in the disposition or temperament of the patient; he becomes irritable, fitful, excited over trifles, takes rebuke badly, becomes quarrelsome, complains of headache and that he is sleeping badly. Sometimes these temperamental peculiarities are the only features, but in other cases the symptoms are of a more pronounced character, with lethargy, somnolence, and delusions. An abnormal feeling of well-being for a day or two before the attack was described by some patients, while others passed into a "dream-state," in which things about them seemed to have lost all reality.

2. The *motor* phenomena consist most commonly of muscular jerks or jumps, sometimes affecting the whole body, at other times only a limb or limbs, of great frequency and occasional marked severity, occurring for a day, or a few hours, before the onset of the seizure. These jerkings lead up to and terminate in a convulsive seizure. These peculiar motor phenomena are also to be found as interparoxysmal symptoms, unaccompanied by a fit, where they would seem to form either series of minor attacks, or exist as paroxysmal substitutes or equivalents.

In cases of serial epilepsy the motor prodromal symptoms may consist of an increasing number of minor epileptic attacks, as may be seen from the charts of this type of the disease; and a similar increase in the minor attacks may be the herald of a major fit, in cases of the combined type to be described later on.

3. *Sensory* prodromata are usually of a subjective character, and consist of paraesthesiae, numbness, pains, a feeling of "pins and needles," etc., in various parts of the body; but considerable importance has recently been placed upon examinations into the objective sensory condition of epileptics by Muskens,[1] of which the following are mentioned.

[1] Muskens, *Arch. für Psychiatrie*, bd. 36, pt. 2, 1902.

This observer found areas of analgesia, mainly corresponding to the segmental areas from the eighth cervical to the fourth dorsal spinal segments: the degree of loss of pain sense varying from slight hypalgesia to complete analgesia. As these zones of anæsthesia were observed prior to a seizure, he regarded them as sensory prodromata of some epileptic attacks.

4. Other prodromal phenomena, which may be described as *somatic*, have been regarded as of much importance by Voisin and his followers, who maintain the toxic, or auto-toxic, theory of the causation of epileptic convulsions. Amongst the more important of these are, furring of the tongue, constipation, flatulence and dyspeptic symptoms, having a definite relation to the onset of the seizures. The pulse may be accelerated, or of low tension.

Under this head may also be placed the various cutaneous eruptions of a vaso-motor kind, which have been found by Féré and others to occasionally precede the onset of the attacks in some cases, *e.g.* pruritus, erythema, urticaria, or angio-neurotic-like rashes and oedemas.

An *explanation of the prodromal symptoms* has been sought for in bio-chemical changes indicative of auto-intoxication; and there are numerous observations upon record which would favour such a hypothesis. Voisin [1] was the first to call attention to the state of urinary hypotoxicity observable before the onset of major fits. Haig [2] directed attention to the fact that there was a diminution in the excretion of uric acid prior to the onset of epileptic seizures; and the observations of Krainsky [3]—of which fuller notice will be taken on a subsequent page—showed that a fall in the uric acid excretion to ·45 gramme was usually followed by an attack.[4] There is, however, no constancy in these phenomena, many epileptic seizures being unaccompanied either by prodromal symptoms or bio-chemical changes of the character above described.

On the other hand, the serial type of epilepsy is most commonly heralded by phenomena such as have been detailed, and, as will be eventually shown, this type presents a clinical

[1] Voisin, *Arch. de Neurologie*, vol xxiii. p. 353. [2] Haig, *Uric Acid*, 1892.

[3] Krainsky, *Mémoires Couronnés*, xv. 1901.

[4] Couvreur (quoted by Binswanger) has shown that uric acid excretion may fall to ·35 gramme with a fit ensuing.

picture comparable to what has been regarded by some as indicative of intoxication.

It is clear, however, that many of the precursory symptoms are of purely nervous origin, and would appear to stand in a relation antecedent to the changes which induce the fit, as do those other well-marked phenomena which succeed it, and are attributable to the passage of the nerve storm over the cerebral cortex (Gowers).

CLINICAL FEATURES OF EPILEPTIC SEIZURES.

In the following account of epileptic fits the method of onset has been used as the basis of description. It is by a study of the aura, or warning, that we are made aware of the part of the cerebral cortex in which the seizure commences. As the warnings of epileptic attacks are referred to most regions and organs of the body, their variety is legion, but the fits, of which they are the commencement, may be collected and described under the following principal categories:

1. Fits with warnings referred to the limbs.
2. Fits with visceral warning.
3. Fits with a warning of head sensations.
4. Fits commencing with psychical warning.
5. Fits with warnings referred to the organs of special sense.
6. Fits without warning.

The relative frequency of the several methods of commencement has been variously stated. Gowers,[1] for example, states that loss of consciousness preceded the first symptoms in two-fifths of his cases, while in three-fifths the patient was aware of the onset of the attacks. Binswanger[2] found an aura present in 31 per cent. of his cases, Herpin[3] in 27 per cent., and Hughes Bennett[4] in 34 per cent. It may be said in general terms that the more sudden the onset of an attack and the more profound the convulsive phenomena and the loss of consciousness, the rarer is the presence of a warning.

1. Fits with Warnings referred to the Limbs.

The periphery of any part of the body may theoretically be the seat of the commencement of an attack, but there are certain

[1] Gowers, *op. cit.*
[2] Binswanger, *op. cit.* p. 187.
[3] Herpin, *op. cit.*
[4] Hughes Bennett quoted by Binswanger.

localities in which the onset is more common. Thus Gowers gives the following in order of frequency: Hand and arm, face, leg and foot, tongue and side of body. Rotation of the head, or of the head and eyes, is also frequent; while an aura referred to the back is only exceptionally noted.

The *aura* may be either unilateral or bilateral. If unilateral, the sensation may be felt in any of the parts above mentioned, and consist of a sensation of numbness or tingling; in others there is a sense of movement, as of twisting or of drawing up of the limb; others speak of sensations of "pins and needles," or of coldness in a limb or of one side of the body. In other cases the sensation is more distinctly cramp-like, especially when the calf of the leg is the seat of the warning.

When the warning is bilateral, similar sensations are described; thus, in one case, mention was made of the warning passing up the body from the feet, or simultaneously from the hands and feet; others referred to a numbness all over the body, while one patient described the aura as a "sensation of something running up the back." Bilateral warnings are also to be seen in "shakes," "jumps," or "starts" in the limbs, preparatory to the onset of the seizure, but these are more probably instances of "petit mal," or minor seizures, rather than true warnings. Of the less common warnings, single instances were observed of "twitchings in the tongue," a "numb sensation in the mouth," and twitching of the lower lip.

In some cases the sensation would appear to pass first up and then down the limb before the convulsive phenomena ensue; and Gowers has referred to instances of sensation descending the limb and re-ascending with the onset of the motor spasm.

The fit. From warnings as above described, the sensation, or cramp, rapidly spreads up the limb, reaches the muscles of the head and neck, which may be thrown into tonic spasm with deviation of the head to the side most convulsed, and consciousness is rapidly lost. The precise moment, however, when consciousness is completely obliterated, varies; but it is commonly stated by patients, that with arrival of the sensation in the head their consciousness is abolished. With the obliteration of consciousness commence the phenomena of the complete epileptic seizure, the tonic spasm and clonic convulsions, which will be described in detail under their proper heading on a future page.

From what has been already said, in the intervals between the complete, or major, fits with peripheral commencement, there may be met *minor attacks* consisting of (*a*) the aura alone: and (*b*) the aura with the initial phenomena of the complete seizure. The minor attacks are therefore identical in locality, warning, symptoms, and march with the onset and initial period of the major fit, and although the seat of onset and its character vary in different persons, it is always, or nearly always, similar in the same person.

Thus we find the minor attacks consisting of a sensation limited to the hand or foot, or a sensation of drawing up of the leg, or of twitching in the tongue (incomplete); while in other cases any of these sensations may pass towards and reach the head, but without inducing complete loss of consciousness, a dazed or giddy feeling only being substituted (complete minor).

2. Fits with Visceral Warning.

Sensations referred to the stomach, or epigastric regions, are the most common in fits having a visceral warning. Less frequent are those in which the aura is referred to the intestines, the lungs, and the heart. Warnings localised in the pelvic organs seem to be of very rare occurrence.

The nature of the *aura* is variable. Some epileptics describe the sensation as that of pain, occasionally of a severe stabbing character, others of a "sensation" in the stomach. One patient said that the warning gave him the impression that his stomach was being "jerked" or "twisted"; others state that the sensation is of a wholly "indescribable" character, while one highly intelligent patient could only describe the warning as a "sense of desolation in the epigastric region." Common terms applied to the epigastric sensations are "spasms," or "cramps," and these are sometimes associated with physical conditions such as borborygmi, flatulence, nausea, vomiting, and a desire to go to stool. The pain is in rare instances referred to the right iliac region, and suggests an affection of the appendix, as was observed in some cases recorded by J. W. Russell.[1] Warnings referable to the heart consist mainly of palpitation, sometimes of pain; while pulmonary auras are recognised by a sensation of suffocation, strangulation, or oppression of breathing. The pain in the region of the heart is stated to resemble in character that of

[1] Russell, *Birmingham Med. Record*, Dec. 1905.

angina pectoris, to such extent that a variety of minor seizure is designated as "epileptic pseudo-angina pectoris."

The fit. Following upon one or other of these warnings, the spasm takes an ascending course towards the throat and head. When it reaches the pharynx a sense of choking is experienced, and the saliva collects in the mouth from pharyngeal constriction. At this period also an arrest of respiration may take place from tonic contraction of the laryngeal muscles. Should consciousness not be abolished already, the patient may call out that he is going to have an attack; or he may sit down, or seek the assistance of anyone near at hand. In one case of this character the initial symptoms were accompanied by a sensation of profound fear and a desire to get away and be alone.[1] The sensation then rapidly passes into the head, and consciousness is abolished, but in some cases not before convulsion has already commenced; sometimes in the muscles of the head and neck, or in the jaws or even in the upper limbs, to be followed by the characteristic phenomena of the complete seizure.

The interparoxysmal phenomena, as in the previous series of cases, are identical with the aura and initial symptoms of the complete attack. Hence in cases with visceral commencement we find the *minor attacks* to consist of (*a*) the visceral aura, and (*b*) the initial symptoms of the complete fit. As examples of these there were observed attacks consisting solely of "an indescribable sensation in the stomach," lasting only a few seconds; and attacks of palpitation, or of difficulty of breathing of a transitory character. In other instances, however, a further development occurred before the symptoms subsided; thus in the patient who had attacks commencing with "something rising from the stomach," a choking feeling in the throat, with a collection of saliva in the mouth, a feeling of dazedness ensued and the seizure terminated, but not before tonic spasm occurred, usually in the left arm; in another, the incomplete attacks consisted of a sensation in the stomach, and the like sensation rising to the head with blurring of consciousness.

The conclusion therefore is the same as previously mentioned, and supports the law of Herpin, that in fits with visceral

[1] Hughlings Jackson (*Med. Times and Gazette*, 1879, and *Brain*, 1899, p. 540) has called attention to *fear*, as a symptom associated with epigastric warning in epileptic paroxysms; sometimes with and at other times without the "dreamy state."

commencement the minor seizures are merely the initial manifestations of the major attacks, which are arrested at a period more or less advanced.

3. Fits with Onset in Head Sensations.

Various cephalic sensations are mentioned as indications of the onset of epileptic fits. The proportion of such cases is, however, not large. The *warnings* are of various kinds, of which the following may be mentioned: pain in the head, sometimes of a severe character and often shooting through the head; others describe a "horrid feeling in the head," "jumping" sensations, or a numb feeling in the head, and, of a more complicated character—"as if something were striking the brain"; while one patient said the sensation seemed as if a "brush was being taken over his brain."

The fit. As already mentioned in the previous descriptions of the march of the spasm, when the sensation reaches the head consciousness is usually abolished; hence in the cases of cephalic warning, it is scarcely likely that the phenomena preceding the loss of consciousness should be of any duration. In most of these cases consciousness is rapidly obliterated, and the phenomena of the complete fit ensue. There may, however, be a turning of the head to one side before this occurs; and cases have been recorded in which an auditory sensation was recorded (Herpin, Gowers) prior to the onset of the major fit.

As one would *a priori* expect, the *incomplete attacks* are rare in this type of case; one patient had the "horrid feeling in the head" with a dazed condition as his minor fits; and another had "jumping" sensations. If present they are usually of the nature of giddy feelings, but even these are rare.

4. Fits commencing with Psychical Warning.

These are warnings of a very special character, and may either occur alone, or be associated, as Hughlings Jackson[1] pointed out, with auras of subjective sensation, such as have been described under epigastric sensations, or more especially with warnings referred to smell and taste. Sometimes the *warning* is of a purely psychical character, such as an intense fear, or "dread of something awful going to happen," or a "terrible feeling of a

[1] Hughlings Jackson, *Brain*, p. 191, 1888, and other papers.

most disagreeable character." At other times the aura may be such as to influence the actions of the patient; thus, in one epileptic the warning was "as if somebody is going to hit me," to escape which she ran away, forming one of the explanations of the so-called "running epilepsy," or *epilepsia cursiva*. In a like fashion an intense sense of fear may lead to striking phenomena, as in a case mentioned by Gowers, where an epileptic put her back against the wall with a look of intense fear upon her face, at the commencement of the fits.

In other cases the warning is of the nature of reminiscence, or "the dreamy state," so well described by Hughlings Jackson in association with subjective sensations of smell and taste.

The dreamy state may consist of a sense of unreality as if surroundings were unfamiliar, or of a sensation that what is happening has been previously experienced. A feeling of fear with an intense desire to be alone has already been mentioned. Probably allied in character to the above are those forms of warning in which objects (visual) or voices (auditory) appear to be fading away into the distance.

The fit. Epileptics with fits beginning with warnings, such as have been described, usually rapidly lose consciousness and pass into the complete epileptic seizure with its tonic and clonic convulsions.

In many cases the *incomplete attacks* are the aura or warning of the complete seizure. One of my patients had as his minor fits the aura of "a horrible sensation," while in another this consisted of a sense of confusion amongst the surrounding objects and a blurring of consciousness. In a third, the minor seizures consisted, either of "a terrible feeling of a disagreeable character" alone, or this sensation with some obscuration of consciousness, but without convulsion.

The patient is frequently able to predict whether the incomplete or complete seizure is likely to occur from the intensity of the warning sensation. The sensation of a sense of fear, with brief loss of consciousness, associated with its physical counterpart of running away as a means of escape, may form the whole of the attack. "The dreamy state" also may form the incomplete attack. Its occasional association with subjective sensation of taste, unconscious smacking of the lips, and chewing movements, followed by a desire to go to stool,

forms a picture of a condition which has received considerable study.[1]

5. Fits commencing with a Warning of Special Sensation.

A. *Olfactory and gustatory auras.* These are not frequent warnings, but in one case there was described a "smell of spring," which occurred both as the representative of the minor attacks and as the aura of the complete seizure. In the subsequent course of the case the aura was no longer present. The sensation of smell varies from a pleasant and agreeable odour to that of a disgusting and unpleasant kind. In some cases the aura is rather that of flavour (so-called taste) than of smell. In some of the cases with warning of a sense of flavour, consciousness is lost almost immediately; but in others the "flavour" descends towards the throat and leads to the "dreamy" state already described (Herpin). During this state smacking movements of the lips, chewing movements of the jaw, and sometimes the action of spitting have been noticed (Hughlings Jackson). During the dreamy state consciousness is lost if the complete fit is going to follow. In the *incomplete* attacks the seizure terminates with the "dreamy" state. Cases in which the aura alone forms the seizure are not common, although this was so in one of my cases, but here no dreamy state was described.

It is in this connection that reference may be made to a highly interesting group of fits, to which Hughlings Jackson has given the name of "Uncinate group of fits."[2]

This term was applied "on the hypothesis that the discharge-lesions in these cases are made up of some cells of different parts of a region of which the uncinate gyrus is part." These cases are characterised by a crude warning sensation of smell or of taste, or there are movements of chewing, smacking of the lips, etc. Occasionally there is a warning of an epigastric sensation, which may occur along with a crude sensation of smell, or with chewing movements. In many of the attacks, but not in all, there is the dreamy state. The attacks may be slight and incomplete as above described; for example, the slightest minor

[1] The reader is referred to the writings of Dr. Hughlings Jackson for examples of incomplete seizures of this type, *Med. Times and Gazette*, vol. i. 1879; *Brain*, 1888; *ibid.* 1898; *ibid.* 1899.

[2] Hughlings Jackson, *Lancet*, Jany. 14, 1899; and *Brain*, 1898, 1899.

fits may be crude sensations of smell with a dreamy state; or, if more severe, unconsciousness may be superadded. The dreamy state may occur alone, and may be of the nature of "Reminiscence"; and a sensation of fear and of impending death have been recorded as additional psychical phenomena.

Much of the symptomatology of uncinate fits refers specially to the digestive system, the crude sensations of smell and taste, and the epigastric sensation being crude developments of digestive sensations. The symptoms described above may be symptomatic of epilepsy only, but quite a number of cases have now been recorded in which an organic lesion (chiefly tumour) of the temporo-sphenoidal lobe has been found.[1]

B. *Visual aura.* Warnings referable to vision are very frequent, and of several kinds. Perhaps the most frequent are visual sensations such as flashes of light, colours, sparks, stars, etc., before the eyes. Other patients describe "objects receding into or approaching from the distance," and sudden loss of vision. Sometimes the warning is more complex; thus one patient described "green things dancing before my eyes," another "something in front of my eyes turning round like a wheel," and a third "figures dancing." Gowers and others report cases with complex psycho-visual warnings in which less specialised visual sensations were followed by more specialised sensations. The "dreamy state" only rarely accompanies a visual aura (Hughlings Jackson).

This aura is rarely associated with other phenomena, consciousness being quickly lost, but giddiness was an accompaniment of "flashes of light" in one case, and in two others there was a feeling as if the power of the left arm was lost, which sensation passed to the mouth and tongue and arrested speech. Sometimes turning of the head precedes the loss of vision and of consciousness, and the onset of the complete seizure.

Incomplete attacks consisting of visual auras are not infrequent in the forms of flashes of light, stars, etc. It is more common, however, to find the aura followed by loss, or obscuration, of consciousness, with slight spasm as the type of the minor seizure. In two cases the minor fit consisted of the aura with loss of power in the arm and of speech and a blurring of consciousness.

C. *Auditory aura.* Warnings referred to the sense of hearing are much rarer than those of sight, but somewhat less

[1] Cases of Jackson and Beevor, Jackson and Colman, J. Anderson, Sanders, MacLane Hamilton, T. Buzzard, etc.

rare than those of smell (H. Jackson). They may consist of subjective sensations of sound, such as noises, hissing, singing, etc., in the ears; or hallucinations of a more complex character, such as "voices being pitched much higher than natural," or a "confused sound of voices," or "as if voices were fading away into the distance." An aura of a sudden sense of stillness, as if hearing had been quickly obliterated, has also been described, but is rare. This has been found in association with the analogous sudden loss of sight already mentioned. Complex auditory phenomena have been also found as warnings, or as the incomplete attack; thus one patient "heard music" at the commencement of his attacks; while in others more elaborate auditory sensations have been described, such as hearing the voices of certain persons saying things which cannot be recalled after the fit is over (Gowers).

As a rule, in the *major attack* consciousness is rapidly and completely lost, but occasionally a turning of the head towards the side on which the sound is heard precedes the loss of consciousness. Associations of auditory warning and dreamy state are rare.

The *incomplete attacks* are, in the majority of cases, the aura of the complete seizure, with or without some interference with consciousness. Thus noises of various sorts, with or without associated deafness, and some mental dazing, may represent the minor fits. The patient in whom there was an aura of "musical sounds" had this sensation, with blurring of consciousness as his incomplete attack. The warning, as such, only rarely forms the incomplete attack in these cases, and, as mentioned by Gowers, complex auditory auras are usually succeeded by the complete convulsive seizure.

The association of auditory aura with local ear disease does not appear to be common. There was one case with middle ear disease and deafness, in which the aura was "a confused sound of voices"; and there were several others, in which Menière symptoms of old standing were accompanied by attacks of unconsciousness and a fall, in which the diagnosis of epilepsy seemed unmistakable. The co-existence of ear disease with epilepsy was noted in a few cases, but there did not appear to be any connection between them.

In the cases with psychical warning and with aura referred to the special senses, the law of identity established by Herpin

holds good, but the exceptions to it are more numerous than in those cases, whose fits are ushered in by a warning of a peripheral or visceral character. The exceptions are of two kinds:

(*a*) During the course of the disease the aura of the complete attack may disappear, consciousness being suddenly lost, while the incomplete attacks retain the character of the original warning. This was seen in one case in which the warning was of an olfactory character.

(*b*) Instances are recorded by Gowers in which the aura of the attacks in the same person were of a different character. Thus in one case minor attacks began with a visual, and major fits with an auditory, warning. Herpin himself refers to a few instances in which more particularly a visual aura was replaced by one of turning of the head. In one of my cases some of the attacks consisted of a sensation of "pins and needles" in the left hand, arm, and cheek; while others were described as of "something passing over me" of a momentary character. In another the minor attacks were described "as if something was running up the back," with temporary dazing, while the major attacks had a sudden onset, without warning.

6. Fits commencing with Loss of Consciousness.

In this category, which forms about two-thirds of the total cases of epilepsy, there is no warning of the onset of the seizure, a sudden and complete loss of consciousness occurring either immediately before, or synchronously with, the onset of convulsions and the development of the complete attack (*vide* p. 84).

The *incomplete attacks* in this class of seizure are an important and characteristic group. As there is no aura, there is in consequence no incomplete minor seizure without interference with consciousness; but there are varieties of minor seizures with obscuration of consciousness, which form common types of epileptic attacks. Owing to the sudden loss of consciousness, the patient is frequently unaware of the attack, and the after-effects of the fit are slight or absent. Many of the cases present features of a psychical nature, the movements noticed during the attacks being not of a convulsive, but of an automatic or semi-purposive type. On the other hand, the

fit may be unattended by any movements, the patient remaining in a dazed or semi-stuporose state, for a few seconds or a minute or two, with the eyes staring and the pupils dilated; while there is an absence of sensibility to noises or cutaneous stimulation—a type of seizure which may more fitly be described as psychical epilepsy.

A very common form of attack is temporary blurring, or obscuration, of consciousness, with muscular relaxation, so that objects held in the hand are dropped, or conversation is momentarily suspended. To such attacks, if recognised by the patient, the terms "sensation," or "losses of memory," are often applied.

In other attacks there is slight and partial convulsion, such as deviation of the eyes, tremors of the eyelids and face, tonic convulsion of a limb, with slight clonic convulsions in another limb, or the head may be oscillated from side to side.

Many clinical variations of these seizures were observed, of which the following are a few instances: (*a*) Attacks in which the patient turns round and falls without subsequent convulsion; (*b*) deviation of the eyes, a cry, and twitching of the arms; (*c*) slight swaying of the body, flushing of the face, twitching of the left arm and leg; (*d*) turning of the head to the left, tonic spasm of right hand and arm, a faint groaning noise, and flushing of the face; (*e*) a sudden fall forwards on the face without convulsion; (*f*) pallor of the face, a flow of saliva from the mouth, deviation of the head, and fall; (*g*) a sudden cry, drops things if sitting at table, or falls.

The initial *cry* is rare in these attacks, showing a less sudden, and less profound, degree of muscular spasm than in the complete seizures. When it occurs it is less strong and less prolonged. But the *fall* is not uncommon, and usually terminates the seizure. It may be forwards, sideways, or backwards, the direction being usually constant in the same patient. Others fall on their knees, or on their hands and knees. In these attacks the patient usually rises rapidly, there being no after-stage of stupor, as in the complete attacks. The face is more commonly pale than cyanosed, but a preliminary blush may precede the subsequent pallor. Urine is rarely, if ever, voided involuntarily in the attacks characterised mainly by a fall, although it may be in minor attacks with slight tonic convulsion.

From such slighter forms of incomplete attack, attended by loss of consciousness, there may be all degrees of seizure up to the severe and prolonged convulsion of the fully developed fit.

The termination of the incomplete attacks is rapid, the patients returning to their work, play, or meal, which have only been temporarily suspended. I recall the case of a patient who, while running after a ball on the cricket field, suddenly lost consciousness, and fell backwards, but instantly rose and continued to run and returned the ball. If the attack has been more severe, the return to complete consciousness is only suspended for a brief interval. There is no stage of stupor such as is seen after the complete attacks, although the patient may be slightly dazed for a time.

Incomplete attacks belonging to this series are frequently succeeded by a somnambulistic-like stage, or a stage of *automatism*. Sometimes there is violence, the patient striking or injuring those about him; on the other hand, he may carry out certain purposive movements, such as taking off his clothes, or emptying out his pockets. One of the cases invariably took out his watch; and another would crouch underneath the table. Many similar and realistic actions have been observed, and described, by writers as sequelæ of incomplete attacks.

The incomplete attack is usually short, a few seconds or a minute or two; but the post-paroxysmal automatism may last from five to ten minutes or more.

The clinical importance of the incomplete seizures is great, as numbers of epileptics suffer from them without their nature being recognised. The disease frequently commences with minor attacks, and may last for years before a complete fit indicates the true character of the malady. Herpin's axiom, that if epilepsy commences with incomplete attacks it usually terminates in a similar way, has been amply corroborated. As a result of treatment the complete seizures are more readily held in abeyance than the incomplete. In many epileptics small series of incomplete attacks herald the approach of a complete fit.

The Complete Epileptic Fit.

Beginning in one of the ways already described—either with a warning, or with sudden and complete loss of conscious-

ness, the phenomena characteristic of the fully developed epileptic seizure are observed. These are, the tonic, followed by the clonic stage of convulsion and the after-stage of stupor and exhaustion.

(*a*) *Stage of tonic spasm.* This consists of a sudden and generalised tonic spasm of the whole of the voluntary musculature. The commencement of this period is frequently ushered in by the "epileptic cry," due, as explained by Gowers, to the tonic spasm of the thoracic and abdominal muscles forcing air suddenly through the glottic chink, narrowed by spasm of the laryngeal muscles. The head is deviated to one or other side, or backwards, and rotation of the whole body may take place, sometimes several times. The face becomes livid and cyanosed, and there is arrest of the respiratory movements from fixation of the chest. The corneal reflex is abolished and the pupils dilate and are inactive. The body may be fixed in a position of opisthotonos, if the spasm is generalised and severe. The upper limbs become distorted, flexed or adducted, the fingers tightly clenched, and the thumbs flexed and adducted into the palms. The lower limbs are fixed in a position of extension and rotation, with the thighs abducted, the feet being usually inverted. The urine is voided from spasm of the bladder musculature.

The tonic spasm may be symmetrical, but usually one side is affected more than the other. Thus, at the commencement of the attack the head and eyes are deviated to one side, and the patient rolls in the direction of such deviation; the spasm may be more pronounced in the limbs on the one side than on the other, and the face is drawn to one side.

(*b*) *Stage of clonic convulsion.* The tonic stage in complete seizures lasts about half a minute, and gradually gives place to shock-like muscular contractions, which assume a clonic character throughout the body, implicating first the extremities, then the body, and finally the head and neck. In this way the head, body, and limbs may be violently jerked, the tongue may be bitten from being jerked between the jaws, the spasm of the respiratory muscles gives place to stertorous breathing, and frothy saliva is expelled from the mouth. As the clonic stage advances the muscular relaxation becomes more marked, and the clonic spasms more intense; these, however, gradually subside by prolongation of the intervals, and finally cease.

During this stage the head may be forcibly banged on the floor, as well as the body and limbs; and the eyes rolled in all directions. Contractions of the intestinal musculature may lead to passage of the faeces, while the seminal vesicles may also empty themselves. Tongue-biting is characteristic of this stage.

It is during this stage that various accidents occur, from the excessive contraction of different muscular groups: such are dislocation of the shoulder, fracture of the teeth and occasionally of the long bones, and rupture of muscles. Haemorrhagic ecchimoses into the conjunctiva may also occur.

The duration of this stage in the completely developed fit varies from one to three minutes.

(*c*) *The after-stage.* After the convulsive phenomena have ceased, the patient remains for a short time in a state of deep unconsciousness. The breathing is quiet and regular, the muscles are flaccid, and the appearance is that of deep sleep. Some cases recover rapidly, and go about their work; the majority, after a temporary wakening, fall into a sleep which lasts for some hours, from which they may awake without any knowledge of the storm, which has so recently overtaken them. On the other hand, severe headache may continue for some hours, or for the remainder of the day. In other cases the phenomena which will be described in their proper places are observed, viz. excitement, violence, and acute mental disturbances; prolongation of the after-stage in the form of acute dementia, or attacks of automatism, which, however, are more common after minor seizures, as already mentioned.

It is generally held that a convulsive epileptic seizure is followed by some increase in the *body temperature.* The extent of the rise, however, depends mainly upon the character and combination of the fits. The greatest elevation is seen in the status epilepticus and in serial epilepsy, where extensive muscular contraction may be largely accountable for the increase. But in single convulsive seizures, minor attacks and fits of a psychical character, an elevation of temperature up to 100° F. has also been observed (Witkowski, Spratling). The only possible explanation of such a rise, in fits unaccompanied by muscular contraction, would seem to lie in an interference with the so-called "heat centres." On the other hand, Spratling has shown that single convulsive attacks and minor seizures may be followed by a

subnormal temperature, which may be explained by the coexistence of a general asthenic state and impaired vitality, as a clinical feature.

The *pulse* also during the epileptic attack has been found to vary by different observers. During the stage of tonic spasm it is usually small and slow, while its complete temporary disappearance has been noted by some writers.[1] During this stage there is sometimes considerable acceleration. On the other hand, others have been unable to discern any material change in its character.

The immediate sequelæ of epileptic fits.

In the train of an epileptic fit, more particularly of the complete attack, but also in the incomplete seizures characterised by some degree of convulsion, changes in the reflex, motor, and sensory functions are observed.

1. *The reflexes.* (*a*) The knee-jerks. During the comatose period the knee-jerks may be temporarily abolished, a condition which is dependent mainly upon the degree of coma and exhaustion. Beevor's[2] observations showed that after the fit the knee-jerks may remain in abeyance for a time, or be increased in force, and that the latter state was more common than the former. In the prolonged coma following upon serial attacks and the status epilepticus, the tendon-jerks may be abolished for several days. This negative state, however, gives place to one of subsequent exaggeration. The state of the jerks immediately after a fit, therefore, depends upon the depth of coma and the degree of the lessening of the muscular tonus. After ordinary fits the jerks are commonly exaggerated, while in status their return is considerably delayed. In close association with the knee-jerks is the ankle clonus. This may be obtained bilaterally after severe convulsions, and Beevor has recorded its presence on the side on which the convulsion has been more marked, as indicated by the deviation of the head. The knee-jerks during the interparoxysmal periods of epileptics are exaggerated.

(*b*) The plantar reflex. The state of this reflex also varies considerably in accordance with the intensity of the seizure. According to Crouzon,[3] during the attack, extensor response is

[1] A. E. Russell, *Lancet*, 1906, ii. p. 152. [2] Beevor, *Brain*, 1882, p. 56.
[3] Crouzon, *Neurol. Centralb.*, 1901, p. 142.

common, while after the attaek is over, extension of the great toe may be temporarily found in about 17 per cent. of all eases. Sometimes flexor response is eonstant, while in others a flexor response may be found on one side and an extensor upon the other. In the epilepsies associated with infantile eerebral palsy, extension of the great toe is constant upon the side of the paralysis.

Whether a post-paroxysmal flexor, or extensor, plantar response is obtained depends upon the degree of exhaustion of the pyramidal system. Hence extension of the great toe is found after severe fits, the status epileptieus, and unilateral eonvulsions without hemiplegia, while flexion may be noted after incomplete seizures and hysterieal attacks.

Collier[1] points out that the presenee of a eonstant extensor response in cases of old-standing epilepsy may be of great value in the early reeognition of organie eerebral disease, whieh sometimes develops after many years in idiopathie epilepsy.

In harmony with the eondition of the plantar reflex is the state of the abdominal reflexes. These are abolished temporarily after severe eonvulsive seizures, on one or both sides.

(*c*) The pupil. Shortly after the eommeneement of the fit (tonic stage) the pupils rapidly dilate, and remain dilated, and immobile to light, during the whole period of the loss of consciousness. This is generally regarded as one of the most constant signs of the complete epileptic attaek. A preliminary eontraction of the pupil has been deseribed by some observers.

With the return of conseiousness, the pupillary dilatation eeases, and the corneal reflex, whieh has been in abeyanee during the fit, also returns.

2. *Motor paralysis* (so-ealled "Exhaustion paralysis"). Paralysis of movement is found after eonvulsive seizures arising from organie eerebral disease, *e.g.* a convulsive seizure may be sueceeded by hemiplegia of a permanent nature, or the localised eonvulsions of Jacksonian epilepsy may be followed by temporary palsies in the limb or side affeeted by the spasm. So also in idiopathie epilepsy, the fit, or series of fits, may lead to a temporary and transient loss, or impairment of motor power, either on one or both sides, but more espeeially upon the side whieh has shown the greater convulsion. These are the so-called post-paroxysmal, or "exhaustion" paralyses, whieh have been deseribed by writers

[1] *Brain*, 1899, p. 88.

upon epilepsy.[1] A variety of post-paroxysmal hemiplegia is not infrequently observed in those who have, in earlier years, been the victims of an infantile cerebral paralysis, which may have to a large extent passed away before the onset of epileptic fits, but which is again brought into prominence by the recurring convulsions of this disease.

The nature of the paralyses following epileptic seizures has long been a moot point, and whether they are to be ascribed to post-paroxysmal exhaustion of the cortical centres (Hughlings Jackson) or to inhibition of the motor functions, is a question to which no definite answer has yet been given; a point in favour of the latter view being the occasional replacement of a fit by partial paralysis, in succession to an aura of a purely sensory nature.

This statement, which is made on the authority of Gowers, who quotes several illustrative instances, would require further corroboration. It is doubtful whether they exist in cases uncomplicated by organic disease. The existence of such temporary inhibition paralysis, without previous motor spasm, is denied by Spratling and P. Clark.[2]

Féré[3] has made an extensive series of dynamo-metric observations upon the motor power after convulsions, and has shown that a considerable enfeeblement of the musculature followed diverse types of attack. After complete seizures there was weakness, at least on one side, in all, or nearly all, his cases; while some weakness was also observed after incomplete attacks characterised by a fall and loss of consciousness. The motor enfeeblement was most pronounced after the attack, and gradually passed away within an hour or two.

Apart from such fleeting and slight paralysis as just described, more pronounced and less transient hemiplegia, monoplegia, and even aphasia may be met with after epileptic attacks. The speech defects consist of hesitation, thickness and slurring of the articulation, having many features analogous to that of paretic dementia. According to Féré they are due to the motor disturbances of the tongue, which is sometimes paralysed for an appreciable period after a fit. These symptoms may last several hours after the attack, and are more pronounced the severer the seizure and the more frequent their repetition.

[1] It is the "epileptic hemiplegia" described by Todd.

[2] Quoted by Spratling, *op. cit.* p. 254. [3] Féré, *op cit.* p. 158.

The true post-paroxysmal palsies occur in limbs which, before the seizure, were intact as regards their motor functions, and last from several minutes to a few hours and then completely disappear.

Binswanger[1] refers to a further type of case in which, during the course of confirmed epilepsy, the seizures were followed by transitory paralysis at first, but which, during the subsequent course of the disease, became a permanent feature of the interparoxysmal periods. These he regards as transitional cases between functional and organic epilepsy, for miliary lesions were discovered pathologically as the exciting cause of the affection.

Sometimes the eye muscles are implicated so as to cause conjugative deviation and strabismus as described by Beevor.[2]

Nystagmus also is described by Féré as a not infrequent phenomenon, the movements sometimes coinciding with the direction of the deviation of the head and eyes during the attack, while in others it is in the opposite direction.

Other motor phenomena following fits are seen in transient contracture and tremor, more noticeable on the side which has been specially convulsed.

The whole question of the causation of the post-convulsive paralyses is shrouded in difficulty, and many explanations of their occurrence have been advanced. That a mono- or hemiplegic paralysis may follow the prolonged clonic convulsions in Jacksonian epilepsy is well known; as also is a temporary increase in the paralysis associated with organic epilepsy, where the convulsions are more marked upon the side of the original motor weakness. Whether an explanation of the diplegic and hemiplegic paralyses, which are seen to follow some idiopathic epileptic seizures, is to be found in exhaustion, or in inhibition, of the motor function is open to argument: one thing is certain, that the co-existent condition of the reflexes—increased tendon-jerks, plantar extensor response, and abolition of the abdominal reflexes—points to a functionally depressed state of the upper motor neurone. I would submit that the changes described in the section on Pathology—small thrombotic occlusions of the cortical blood-vessels—may provide an explanation, not only of the convulsion in predisposed persons, but also of the subsequent temporary paralysis.

[1] Binswanger, *op. cit.* p. 211. [2] Beevor, *Brain*, p. 52, 1882.

The persistence of paralysis for days or weeks, after serial attacks and the status epilepticus, would favour this view, as it is in these conditions that the thrombotic lesions are especially seen.

3. *Sensory paralysis.* In addition to the motor disturbances, important sensory post-paroxysmal phenomena are found in some cases.

(*a*) *Diminution* or *loss of cutaneous sensation*, general or partial in distribution, may exist for many hours or days after an epileptic attack. It is most marked in, or limited to, the limbs affected by transitory motor weakness, but does not stand in any direct relation, either to the intensity of the seizure, or the degree of the motor convulsion (H. Bennett). The loss of sensibility is especially referable to touch and painful impressions (hypaesthesia and hypalgesia). It is not necessarily co-extensive with the motor palsy, being sometimes of a hemiplegic type, when the motor weakness is limited mainly to one limb. In one case of frequently recurring seizures of an incomplete major type, the clonic spasms were mainly observed in the left arm, the left leg being implicated to only a slight extent, yet the hypalgesia affected the whole of the left side to apparently the same degree.

(*b*) *Amblyopia*, or visual defect, has also been observed after epileptic attacks, and more particularly in the form of constriction of the visual fields (Thomsen and Oppenheim). This phenomenon was found by these observers more especially after attacks of a psychical character, rather than as a sequence of complete convulsive fits, and it was not uncommon to find it in association with temporary anaesthesia. Féré, on the other hand, holds that visual defect is not limited to the psychical type of seizure, but is found as a result of all fits with loss of consciousness. Sometimes the restriction is limited to colour vision, particularly for red and green (Pichon, Féré). Blindness has also been noticed in the intervals of attacks of serial epilepsy (Fano), and at the commencement of status epilepticus (Féré). Hemianopic restriction of vision is relatively rare after epileptic fits.

As with motor paralysis, post-paroxysmal visual anaesthesia is of a temporary character, passing away in the course of a few hours.[1]

[1] H. Bennett, quoted by Binswanger, *op. cit.* p. 214.

(*c*) *Deafness* has been described as a rare form of post-paroxysmal palsy. Féré found temporary diminution of the sense of hearing after epileptic attacks in a few cases.

(*d*) Of still rarer occurrence is temporary abolition, or defect, of the senses of *smell* and *taste*. Binswanger refers to a case of prolonged anosmia and ageusia after serial seizures.

4. *Loss of body weight* has been stated to be a common sequence of all epileptic seizures, sometimes to the extent of several pounds (Kowalevsky); but this has not been confirmed by all observers, and it should be noted that the loss is only slight. On the other hand, after serial attacks, and status epilepticus, a marked loss of weight has been observed, from which recovery is usually rapid.

I have observed an extensive and progressive loss of weight in confirmed epileptics, who are subject to frequent seizures, and in whom dementia is advancing. A loss of from fourteen to twenty pounds was noted in several cases during the course of as many months.

CHAPTER V.

THE CLINICAL STUDY OF EPILEPTIC FITS (*continued*).

Clinical types of epilepsy—Major type—Minor type—Combined type—Serial epilepsy—Status epilepticus—Nocturnal and diurnal forms—Senile epilepsy—Onset and course—Long remissions—Termination.

CLINICAL GROUPINGS OF EPILEPTIC FITS—CLINICAL TYPES OF EPILEPSY.

The various ways in which epilepsy manifests itself clinically may be described according to (*a*) the type of the seizures, (*b*) the frequency of the seizures, and (*c*) the time of their occurrence.

It is only in Institutions, where epileptics are observed daily and hourly over prolonged periods, amounting in some cases to many years, and where the fits are regularly charted as regards frequency, severity, and incidence, that true pictures of the clinical varieties of the malady can be accurately and graphically studied.

The clinical types of the disease, to which reference will be made in the following pages, were all studied in this manner, and as complete a record as possible of the fits was obtained on specially constructed charts.

As already defined, there are two leading types of epileptic seizure—the major and the minor fit. Hence there are found two primary clinical groups of the disease—the major and the minor variety, according as the seizures are solely of the major and minor type.

A third and very common group is that characterised by a combination of the major and minor seizures, the so-called combined type.

In the next place, it is important to ascertain the relative

frequency of the three types of epilepsy, and for this purpose a collection of 280 cases of the fully developed disease was studied, all the cases having been under constant observation for periods varying from six months to ten years.

Serial epilepsy is a highly interesting form of the disease, in which the type of seizure may be either the major or the minor variety, and in which the seizures occur in series. This has been separately scheduled, as it forms a variety, or complication, of the confirmed disease, requiring special description.

The numerical frequency and percentage of these groups are stated as follows:

Major type, - -	163 cases	58·2 per cent.
Combined type, -	107 „	38·2 „
Minor type, - -	10 „	3·8 „
Total, -	280 cases	100 per cent.

These figures would appear to accord very closely with those given by Spratling from a much larger number of epileptics. This observer found, amongst 1325 cases of epilepsy—

The major type in - -	60 per cent.
The combined type in - -	32 „
The minor type in - -	5·5 „

From these two series of figures it is obvious that the commonest clinical variety of the disease is the major type, characterised by the recurrence of convulsive seizures; that the minor variety, indicated by the type of seizure already described, is a relatively rare clinical manifestation, but that when the two types of fit are combined to form the clinical picture a frequent group of epilepsies is noted.

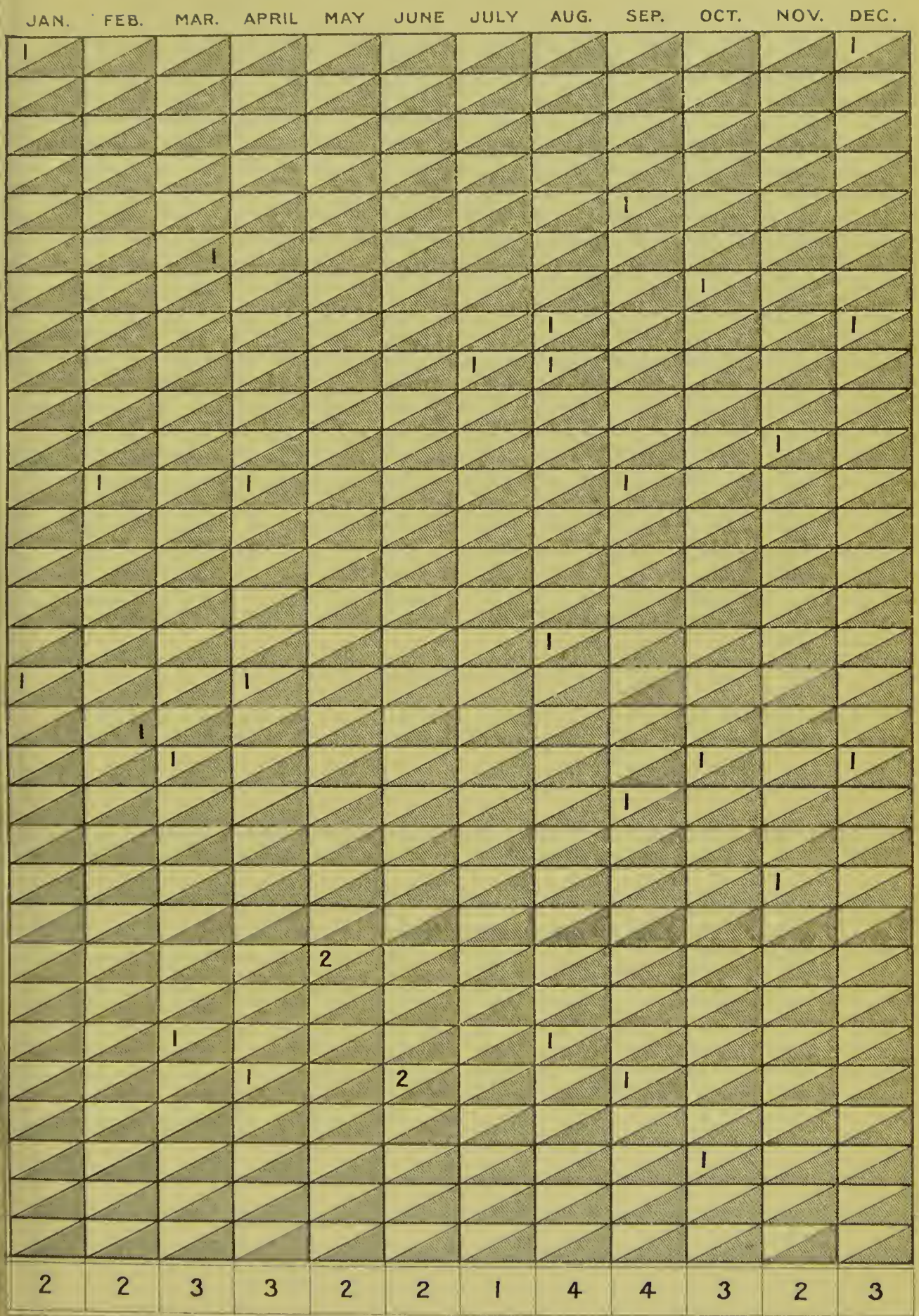
(*a*) *Clinical groupings based upon the character of the seizures.*

1. The Major Type.

This variety of the disease is characterised by the recurrence of convulsive seizures varying in frequency from one or two, or less, up to twenty or more per month. They may occur either by day or by night, or during both the waking and the sleeping hours.

Many cases of this type, as indeed most forms of epilepsy,

An Average Type of Major Epilepsy.



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present a peculiar constancy in the incidence of the seizures. Over many years there is little or no change in the monthly or yearly total. An average weekly, monthly, or yearly frequency having been established early in the course of the disease, little variation takes place, so that charts, noting the incidence of the fits for many years, show a striking similarity. On the other hand, there are cases in which the frequency of the seizures increases as time goes on; but there is rarely any sudden change in a well-established type, unless there arises a complication, such as a serial outburst, or the development of status epilepticus.

The fits may occur singly, or in little groups of two or three; there is no regular periodicity in their incidence, although there is a tendency for the attacks to supervene at fairly regular intervals. In women, at the commencement of the disease, the tendency is towards their recurrence just before or after the catamenial period; but this association is usually lost as the malady becomes chronic.

Three charts are appended showing an average, a nocturnal and a severe form of the major type of epilepsy (Charts 1, 2 and 3).

Chart 1 shows a year's record of fits from an average case of major epilepsy. The fits occur either singly or in couples, more commonly by day. The fit incidence is such, that it may be estimated by the month; the total number of fits for the year under observation amounting to 31. Chart 2 is an eight months' record (Sept., 1895 to April, 1896) of a case of severe major epilepsy, in which the fits recurred so frequently as to be counted by the week, the total for one month amounting to several score. The fits were mainly nocturnal.

These cases have been selected as illustrative types of the major variety of epilepsy, but all gradations are met with from seizures recurring so seldom as once or twice a year, up to one hundred or more per month.

In Chart 3, there is shown a case of nocturnal major epilepsy, with an increasing frequency of fits during the later months. Coincidental with the increased number of seizures, there ensued a deepening of the mental decrepitude.

The mental condition associated with the major type of convulsion is not characterised by any special degree or frequency of dementia: an almost equal percentage of cases are to be

found distributed over the four divisions into which the mental attitude of epileptics has been divided (see p. 149).

The degree of dementia is largely determined by the frequency of the seizures; thus we find that those cases in which the attacks recur as often as once or twice daily, or weekly, are associated with deeper states of dementia than those having only a monthly, or three monthly, fit incidence; while of the rare instances in which the attacks only recurred once or twice a year, or in those in which arrest took place, the mental state remained normal, or was affected to only a minimal extent.

As years pass on, and the frequency of the attacks increases, the mental attitude shows signs of further deterioration; or the accidental occurrence of the status epilepticus may suddenly induce an attack of acute dementia, from which recovery is at best imperfect, or may be the starting point of a prolonged and indefinite period of delusional insanity.

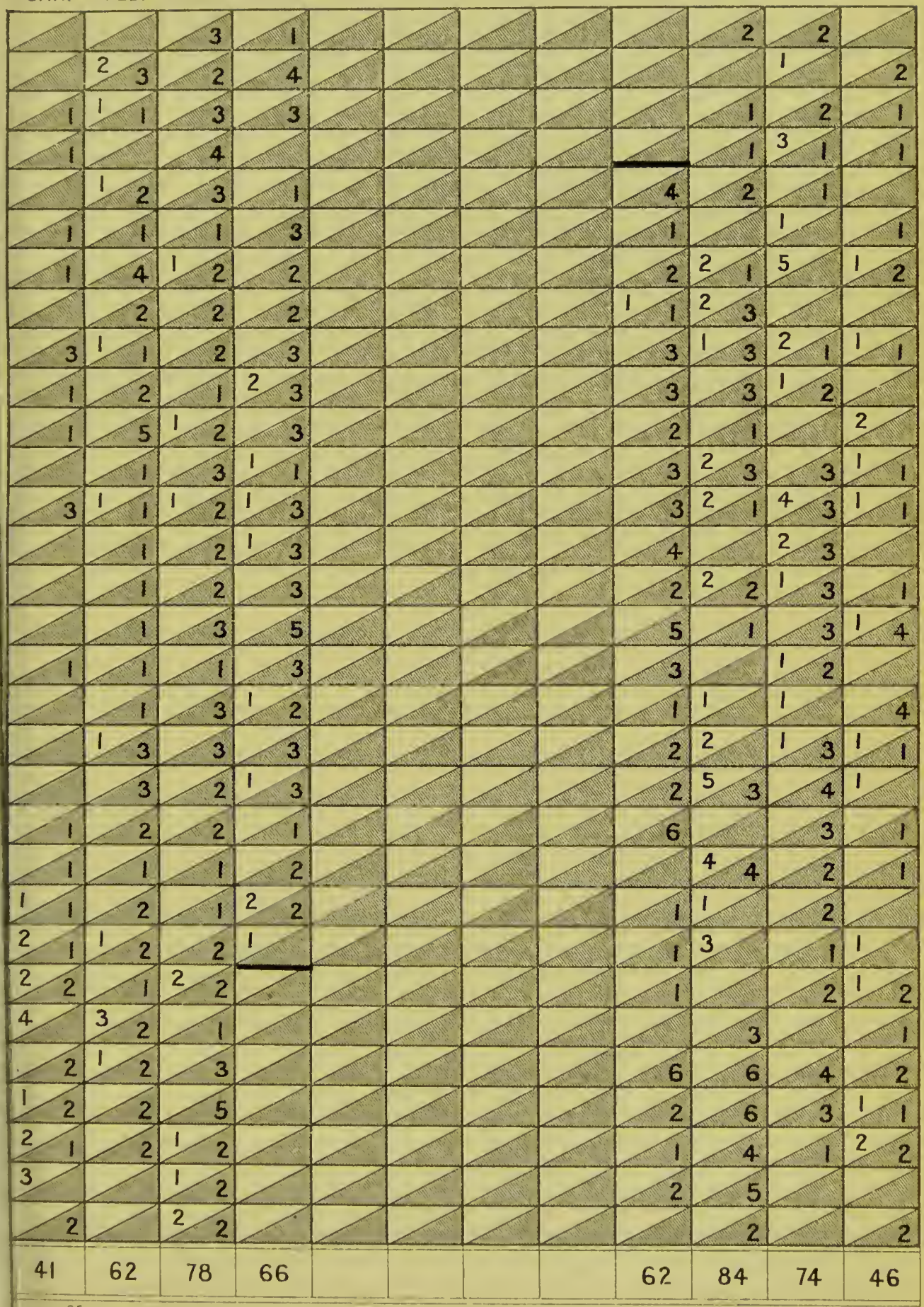
2. The Minor Type.

This is a less frequent type than the preceding, being found in its pure form in only ten cases, or 3·8 per cent., of my series. The type is characterised by the presence of the "petit mal," or minor seizures, the forms of which have been described on a previous page. As a rule, this form of the disease is only encountered during waking hours, and the numerical frequency of the attacks varies as much as the "grand mal" seizures, from several in the course of the month up to eight or ten daily.

As in the major variety, there is a certain constancy of type observed throughout the course of the malady. As time goes on, a tendency to increase of frequency of the seizures may be noted. On the other hand, the minor type, when established, rarely, if ever, changes into the major variety; major fits may ensue later in the course of the disease, but the minor attacks do not disappear, nor are they replaced by the grand mal seizures, the change being from the minor type into the combined form with the minor seizures in excess.

A characteristic instance of this form of the disease is illustrated in Chart 4, which depicts a severe type of minor epilepsy. The seizures consisted of a sudden fall with momentary loss of consciousness, followed by instant recovery.

A Severe Type of Major Epilepsy.



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As the year advanced the "falls" increased rapidly in number, until a condition of status epilepticus ensued on Nov. 22nd. This patient eventually developed delusional insanity, and was removed to an asylum.

The mental attitude of the minor type is more characteristic than in the major type. The usual psychical state consists of a slight degree of mental impairment, chiefly loss of memory, more particularly for recent events. It is rare to find examples of this type with a normal mental state: on the other hand, profound degrees of dementia are less common than in the other groups.

The rarity of the pure type of minor epilepsy is remarkable; it occurs, moreover, in persons endowed with high intellectual abilities.

3. The Combined Type.

This is a common type of epilepsy, being found in 38·2 per cent. of the cases examined. It occurs in three varieties.

(*a*) Major attacks in excess of minor.

(*b*) Minor attacks in excess of major.

(*c*) Psychomotor attacks in association with major fits.

The greatest numerical frequency of seizures amongst the cases of epilepsy, with the exception of some rare cases of serial epilepsy to be immediately described, was found in the combined type, as many as 120 or more fits being recorded per month over prolonged periods. As already mentioned, when the primary types were described, there is a characteristic constancy in the frequency and character-combination of the seizures, the tendency, however, being eventually to an increase in the number of the attacks. In the second sub-group of this type, the infrequent major seizures are usually nocturnal, the minor attacks occurring only during the waking hours.

The third sub-group, as above mentioned, is a frequent type of character-combination, in which numerous attacks of a psychical or psychomotor form, may occur in combination with an occasional major seizure. The relation of the psychical attacks to the convulsive fit is usually constant, in that a short series of these attacks may lead up to the convulsive seizure, after which there is freedom for a time. These are usually observed at or about the catamenial period. In other cases one finds that if the convulsive seizures temporarily diminish in

frequeney, the minor and psyehomotor attacks usually become more frequent. In other patients the convulsive attack, instead of "elearing the air," as it were, induces a tendency to the oceurrenee of minor seizures, so that instead of leading up to the major fit, as just mentioned, they are observed to sueeeed it, a phenomenon whieh has its eounterpart in the automatism, whieh is noted as a post-paroxysmal psyehosis.

There may also be mentioned in this relation those "commotions," jerks, jumps, or "shakes," whieh, oeeurring in small series, often lead up to, and terminate in, a major convulsive attaek. This last is not an uneommon type of the eombined group of fits.

The eombined type is illustrated by two eases, reeorded on eharts 5 and 6. Chart 5 is an example of a common variety of the eombined type, the major seizures being in exeess and of noeturnal oeeurrenee, while the minor are observed during waking hours. It will be noted that when the major fits are more numerous, the minor are in abeyanee, and *vice versa*. The major attaeks sometimes oeeur in small series.

Chart 6 depiets a not uneommon form of the eombined type, in whieh small series of minor, or psyehomotor seizures, precede the onset of and lead up to the major explosion. The minor, on the other hand, may oecur without the major fit.

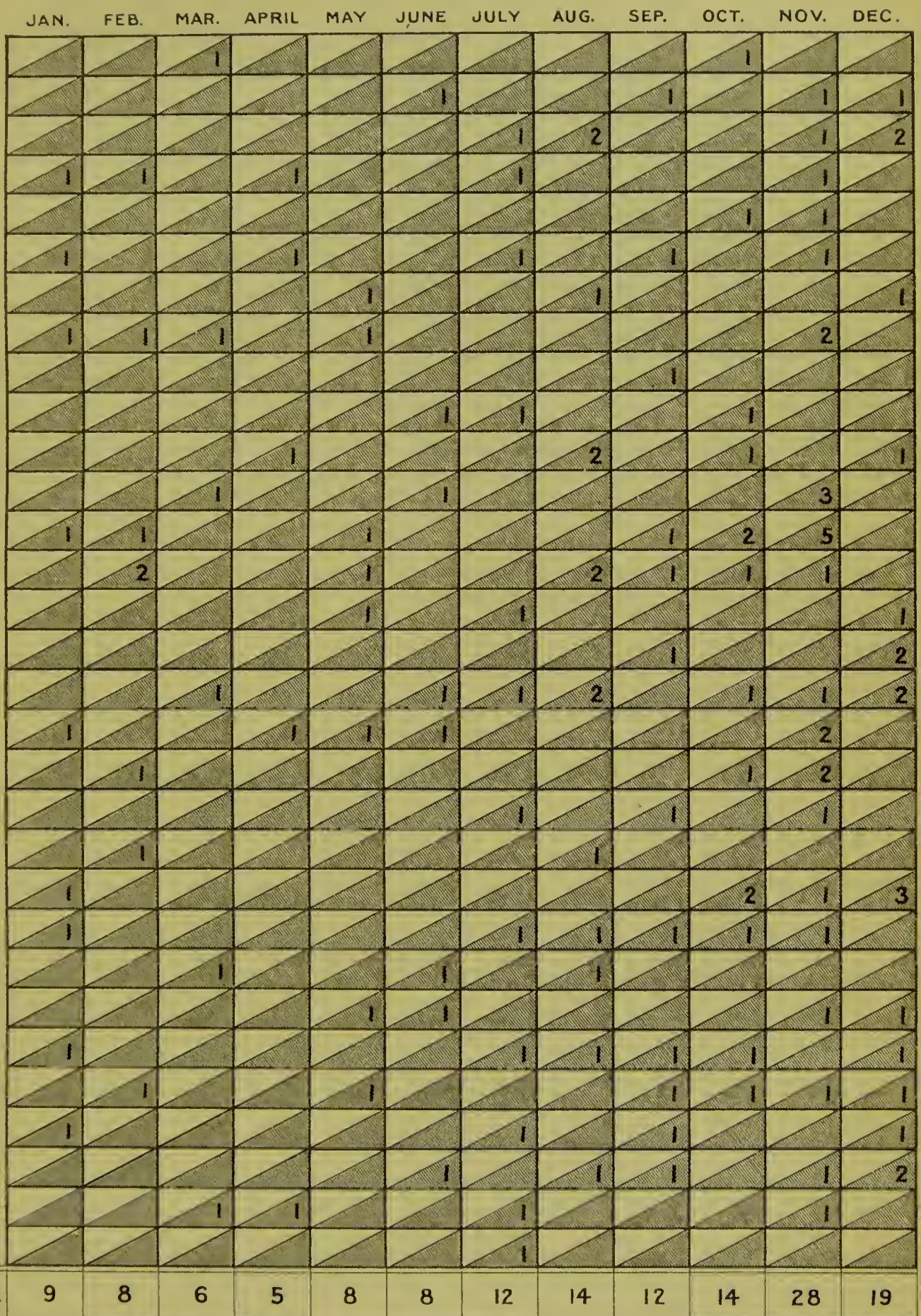
As might indeed be expeeted from the faet that this is the severest persistent form of epilepsy, the deepest degrees of dementia are found in assoeiation with the eombination of the major and minor attaeks. Out of a total of 71 cases (p. 149) in whieh the mental condition was investigated, 37 per eent. were elassified as of normal and slightly defieient mental attitude, while the remainder—63·2 per eent.—presented the severer degrees of dementia. It is the type of the disease least influeneed by treatment, and it is that one in whieh the stigmata of degeneration are most often seen.

(*b*) *Clinical groupings according to the frequency of the seizures.*

1. Serial Epilepsy.

This is a speeialised and interesting variety of epilepsy, in whieh a sueeession, or series, of paroxysms, sometimes of the minor, but more freqnently of the major type, follow eaeh other with greater or less rapidity over a limited interval of time.

CHART 3. AN AVERAGE TYPE OF MAJOR NOCTURNAL EPILEPSY.



To face page 98

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It may be described as a sub-acute form of epilepsy, in that the seizures, instead of recurring singly, or in groups of two or three at a time, recur in batches up to a dozen or more, over several days. It differs in its typical form from the status epilepticus by the fact, that consciousness is regained to a greater or less extent between the seizures, but resembles the status, in that there are usually well-marked prodromal symptoms, sometimes of the usual psychical character, as already elsewhere described; and by the further fact, that a series is followed by the phenomena usually observed after severe major fits, but of a more intense and protracted character.

It is found, first, as the type of the disease in a number of cases, my statistics showing that this occurred in 38 cases out of a series of 280, or 13·5 per cent. Secondly, as a modified form of status epilepticus, in cases of old standing major epilepsy, or in cases in which the seizures are assuming an increasing frequency. It is, however, impossible to draw a hard and fast line between these two varieties, as instances of serial epilepsy are found in which, every now and again, the series is prolonged into a characteristic, but mild, type of the status epilepticus. This latter condition, indeed, is shown in some of its most characteristic forms in cases of serial epilepsy. The difference, therefore, between seizures occurring singly, in twos or threes, in series of a dozen or a score, and the classical status epilepticus, is only one of degree. I would, in consequence, *define serial epilepsy as a sub-acute form of epilepsy, or a mild and modified status epilepticus.*

In the type of the disease characterised by short serial outbursts (true serial epilepsy), series of major fits are more common than series of minor attacks; but there is also a variety of serial epilepsy, in which serial periods occur in combination with single fits, either of the major or minor type.

Two cases of serial epilepsy are illustrated on charts 7 and 8. Chart 7 is an average case, the fits being of the major type, and recurring in series of about a score every alternate month. They occur both by day and by night. In the intervals single seizures, or an aborted series, may be seen. This is a highly special type of epilepsy of not uncommon occurrence.

In chart 8 a severe type of serial epilepsy is depicted. In

this case the general type is that of serial outbursts of a severe form, the attacks on some occasions being so frequent as to constitute the status epilepticus. It may be held that this is not a true case of serial epilepsy but merely a severe form of the major type. In this connection it may be stated that the disease, as illustrated, only persisted for about a year, and was associated with profound dementia. Eventually marked improvement ensued both in the number of the fits and in the mental condition.

A serial outburst may be heralded by an increase in the number of single seizures, which may recur from time to time. The attacks increase in frequency until a summit is reached, and then gradually diminish. The after-period is characterised by an almost complete temporary cessation of fits, though an occasional single seizure may sporadically appear. From the maximum of a dozen of fits or more, there are all gradations down to four or five attacks constituting a series.

The duration of the serial period may be from a few hours to three or four days. The fits occur either during waking or sleeping, or both. There were some instances in which the series commenced with a major seizure, which was succeeded during the following day or two by frequent minor attacks. During the series the temperature may be slightly elevated, but this is rare; and the after-stage is characterised by temporary lethargy, which gives place to transient delusional conditions requiring care and attention.

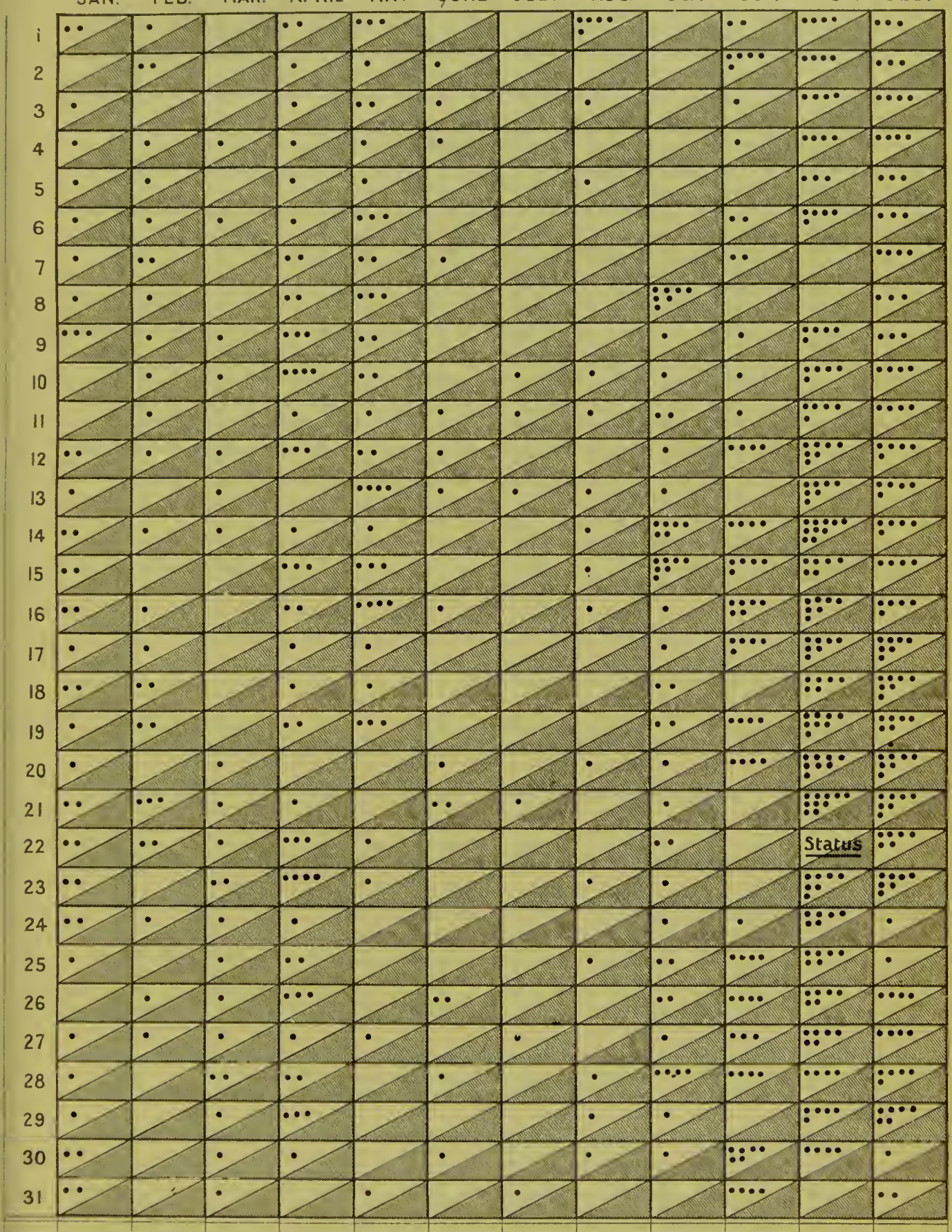
This type of the disease leads to more serious temporary mental failure than any other. After the short series, the post-paroxysmal mental phenomena of acute dementia and stupor repeatedly occur, and in time induce dementia of a persistent type. After the more prolonged series, the acute dementia has been seen to pass directly into a sub-chronic or chronic form, from which recovery rarely takes place. On the other hand, the recovery from the mental enfeeblement, induced by some prolonged and severe series, is occasionally surprising, some alteration in character, such as increased irritability, being the only feature which strikes the observer.

Out of a total of thirteen cases of serial epilepsy, considerably more than one-half (70 per cent.) showed the severer grades of dementia.

Some facts will be mentioned and arguments brought forward

CHART 4.

A Severe Type of Minor Epilepsy.



To face page 100.

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in a later chapter to show, that serial epilepsy, and the status epilepticus, may be the forms of the disease, which have an exciting cause in auto-intoxication.

2. Status Epilepticus.

Status Epilepticus (*état de mal* of the French writers) is the most acute manifestation of epilepsy, and in some of its developments may form an acute type of the disease. It has been defined by Clark and Prout[1] as "the maximum development of epilepsy." It is a temporary state, occurring during the course of the disease, in which one fit follows another so closely that the comatose after-stage gives place to the succeeding convulsion without a return to consciousness. It may therefore be regarded as a more complete convulsive paroxysmal phenomenon than serial epilepsy, which latter has indeed been considered and described as a modified form of the status epilepticus.

The close relation which exists between serial epileptic periods and the status epilepticus may be seen from the following facts:

(*a*) A serial period may be replaced by, or develop into, a status period in which death may occur.

(*b*) Single attacks may, as the disease progresses, pass into small serial periods, and these again into status periods.

(*c*) The serial period, if severe, may be accompanied by the general phenomena of the status period—elevation of temperature with acceleration of pulse and respiration.

(*d*) The after-stage of the two conditions is frequently alike, viz., acute temporary dementia and stupor, resolving through delusional states, irritability and restlessness, into the average mental condition before the series.

In my series of 280 cases observed for periods of from two to ten years, 15 patients, or 5 per cent., showed at one time or another a typical development of status epilepticus. In these fifteen cases there were 29 attacks of status, as shown by the following table:

In 1 case,	7	status periods	in 7	years.
In 1 case,	4	„	„ 2	„
In 1 case,	3	„	„ 2	„
In 3 cases,	2	„	„ 2	„
In 9 cases,	1	„	„ during observation.	

[1] Clark and Prout, *American Journal of Insanity*, Oct. 1903, where an extensive bibliography will also be found.

A review of the cases, which exhibited the status epilepticus, revealed the following facts as showing its relation to the confirmed disease:

1. Status epilepticus may occur as the type of the disease. This was shown in one patient who had an attack of status each year for seven consecutive years, in the intervals of which there were single seizures of infrequent occurrence. This would appear to be the most acute type of confirmed epilepsy.

2. As an occasional acute development in cases of the severe combined type, more especially when this presents the combination of short series with single fits.

3. As the ultimate issue in old-standing cases of confirmed epilepsy, single fits developing occasionally into short series, which may from time to time increase into a status period.

4. As the result of an accidental circumstance during the course of the disease, such as sudden stoppage of bromide medication, a strong emotional or psychical excitement, the puerperium, an acute inflammatory disorder, and a fall or blow upon the head.

The status epilepticus is found in both the major and minor types of epilepsy, more commonly the former. It is also seen, as already mentioned, in the combined type. It has to be carefully distinguished from severe and frequently recurring epileptic seizures.[1]

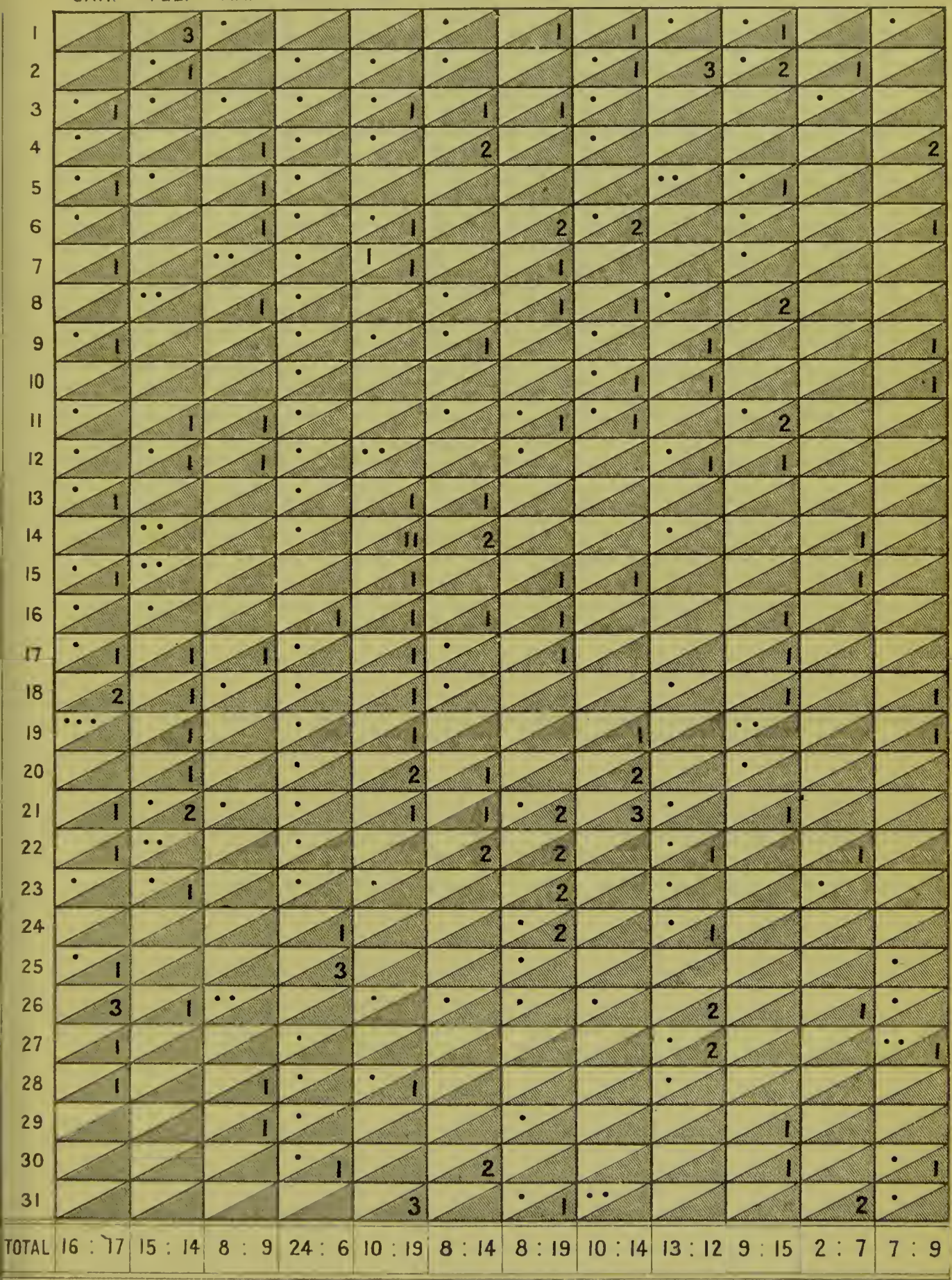
The onset of status is usually foreshadowed by an increase in the number of seizures of the customary type, as is also seen in the corresponding condition of serial epilepsy; on the other hand, some status periods present a sudden onset, which are, according to Clark, almost invariably due to arrest of sedative treatment, although this is by no means the only cause of suddenly developed status.

With the development of the status period convulsive attacks begin to follow each other with considerable rapidity, each attack being complete with its after-stage, consciousness being at first regained; but as the interparoxysmal periods shorten, the comatose stage is prolonged, and the seizures, increasing in

[1] The number of fits that may occur in any given time in epilepsy is a matter of some interest. Clark refers to a case of Leroy's in which there were 488 fits in twenty-four hours, and 1000 in three days; and to one of Parson's with 1400 attacks in four weeks. I have myself seen 2080 in eight weeks, 673 in ten days, 820 in five days, and as many as 289 in twenty-four hours.

CHART 5.

An Illustration of the Combined Type of Epilepsy.



To face page 102.

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frequency, eventually induce a state of continued clonic spasm, the final convulsions of the preceding seizure having scarcely died away before those heralding the approach of the next commence. From this, which may be looked upon as the apex of the period, the spasms begin to diminish in frequency, so that towards the termination of this stage single isolated spasms may be the sole representatives of the paroxysms. Now ensues the convulsionless after-stage of continued coma, stupor, and exhaustion, in which the patient may die. This stage is characterised by the usual phenomena of coma from other causes; stertorous breathing, a cold clammy skin, the involuntary voiding of urine and foeces, abolition of the tendon reflexes, an extensor plantar reflex, elevation of temperature, sometimes to hyperpyrexia and increased pulse rate. Occasionally during this stage, isolated convulsive paroxysms occur.

If recovery is going to take place, the coma gradually lessens in intensity, giving way to a state of stupor, from which the patient may be temporarily aroused. This eventually passes into a delusional and hallucinatory period, with restlessness and irritability, and recovery more or less complete ensues in from one week to ten days.

On the other hand, status epilepticus may be fatal in the early part of the comatose stage, death taking place usually from exhaustion, with symptoms of a typhoid state, and sometimes acute decubitus.

The duration of the status epilepticus is important, as many instances of serial attacks, which are really modified forms of status, present features comparable to what has just been described. According to Clark and Prout, a status period does not last beyond three or four days, usually only about eight or ten hours.

This definition would appear to restrict the status epilepticus too severely to outbursts of frequently recurring attacks over short periods. It is true that some of the most acute forms of status, in which death may rapidly occur, only last a few hours (chart 9); but there are other outbursts, in which fits recur with at first increasing and then diminishing frequency over much longer periods, a condition which should, in my opinion, be regarded as a more prolonged, or sub-acute, variety of the status epilepticus (chart 10). It will be therefore seen that the status epilepticus may vary from short and severe convulsive

outbursts, lasting eight, ten, twelve, or twenty hours, up to ten or eleven days. That the difference between these types is merely one of degree and intensity of symptoms, is obvious from the fact that the prolonged phases present all the characteristic phenomena of the short outbursts in a modified way; and that death may occur during the comatose after-period of the prolonged status, as in that following the shorter attacks. Hence the number of fits constituting a status period varies from a dozen or more in a few hours (chart 9) up to several hundred in a few days. In one case (chart 10) 564 fits were recorded in twelve days.

A condition similar to the above, and allied to it, is the *status hystericus*, occurring in subjects whose recurring paroxysms are of an hysterical character.

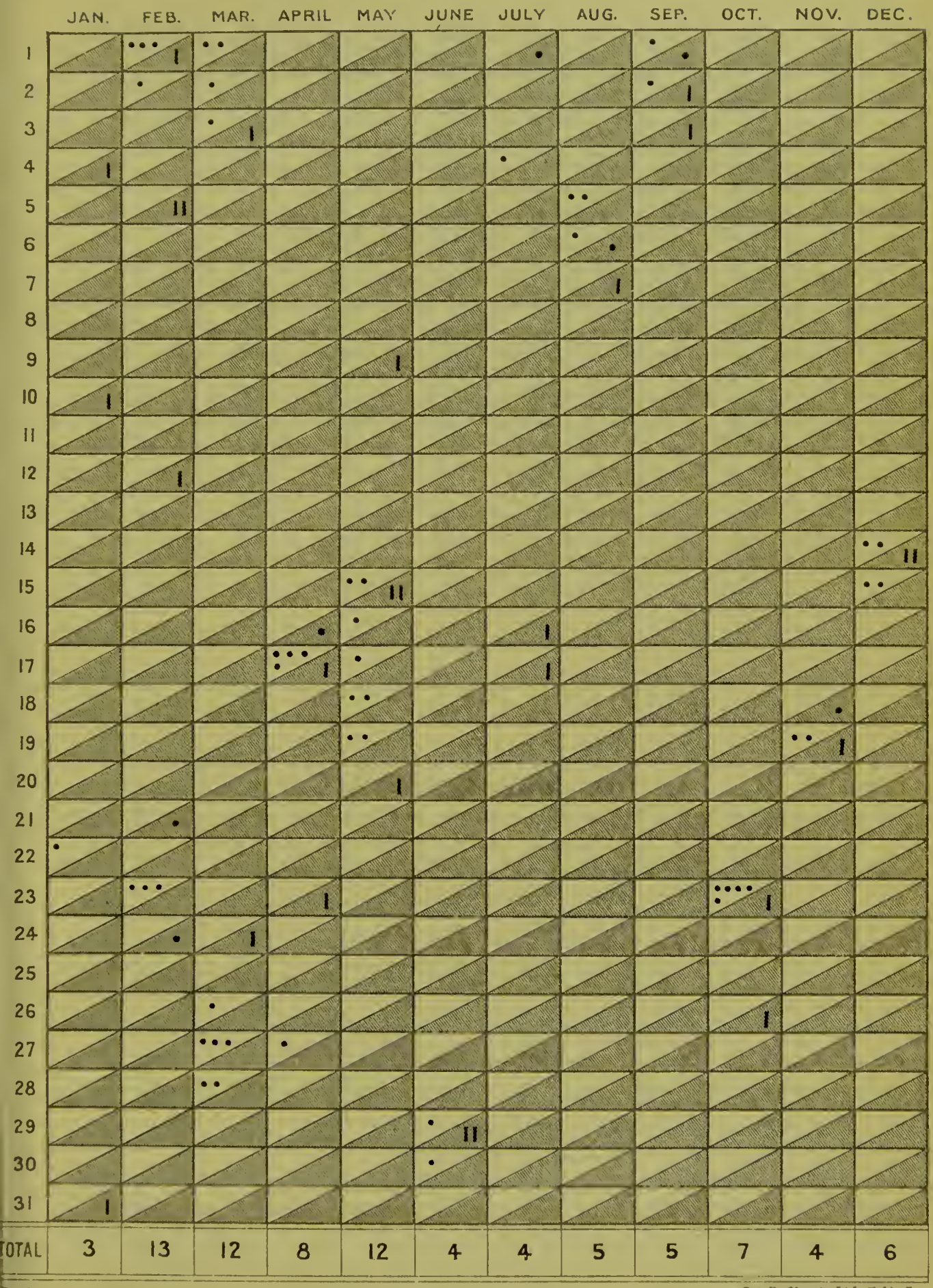
Many epileptics never develop status epilepticus, but it is undoubtedly more frequent than is generally supposed. As already shown, it may in rare cases form the clinical type of the idiopathic disease; it may be an accidental circumstance, the result of controllable or uncontrollable causes; and it is not uncommon in organic epilepsy, where the convulsions are usually limited to the paralysed limbs (status epilepticus unilateralis) and in general paralysis of the insane.

Some other acute convulsive conditions are no doubt instances of the status epilepticus; and as illustrations of this may be mentioned the prolonged convulsive attacks which usher in the onset of infantile hemiplegia, some of the so-called eclampsias of the puerperium, and those rarer forms of convulsion which accompany the development of hemiplegia in elderly persons (Clark and Prout).

Although the status epilepticus is the natural termination of the disease in some cases, it may, as already mentioned, be accidentally induced by various circumstances. Nor is its incidence limited to one attack. Just as we have seen recurring periods of serial epilepsy, so there are observed recurring attacks of the status epilepticus, up to as many as seven in one of my patients, and nine in a case of Clark and Prout's.

The prognosis is grave, but not necessarily fatal. The statistics of death from status epilepticus vary enormously; but there would appear to be a tendency to a great diminution in the mortality both during and after the status, no doubt from the more general recognition of the condition and

CHART 6. THE COMBINED TYPE; PSYCHO-MOTOR AND MAJOR FITS IN SERIES.



To Face page 104.

R. & R. Clark, Ltd. Edin.r

its mode of onset, as well as from the careful treatment which the victims reeeive in Institutions for epileptics. Binswanger gives a mortality of 50 per cent.; Lorenz, 45 per eent.; Clark and Prout, 33 per eent. My own figures are: Three deaths in twenty-eight attaeks of the status epileptieus, or 10·7 per cent.

3. A Type of Epilepsy characterised by a Few Fits only.

There would appear to be a clinieal form of the disease, eonstituted by a few seizures oeeurring over a limited period of time. These eases are rare, but are probably more numerous than the reeords show, as many of them never eome under observation or treatment at all; but investigation of the family histories of epilepties oeeasionally reveals the fact, that some one member had a fit or two at one period of life, of whieh there has been no reeurrenee. It is not unlikely that some eases of epilepsy cured by treatment are really of this eharaeter. In my series I find a few instanees of this type, as follows:

Case 1—2 fits when aet. 14 years,							No more	10	years later.
„ 2—6	„	16	„				„	10	„
„ 3—2	„	19	„				„	11	„
„ 4—3 fits between 18 and 19 years,							„	15	„
„ 5—Fits from 28-32 years,							„	17	„
„ 6—Fits from 28-32	„						„	22	„
„ 7—Fits from 16-20	„						„	40	„

It may be argued that these are not really cases of idiopathie epilepsy, but of eonvulsions, due to some temporary exeiting cause. This argument, however, is seareely tenable for all the eases, as three of them had seizures reeurring during a period of four years; and it has been already shown that there is an aeute epilepsy, which may, or may not, be followed by the disease in a ehronie form. I hold then that these are instanees of a type of epilepsy whieh requires reeognition; they have an important bearing on heredity, as is shown in the faet that a parent who has had only one eonvulsive attaek in a lifetime, may beget a ehild who eventually beeomes an epileptie. This would appear to be eomparable to what is oeeasionally noted in the family history of epileptics, that one or other parent had a fit at the onset of the paralytie attaek whieh eaused death. A convulsive seizure in a parent, either in late life at the onset of a stroke, or in the earlier years about puberty,

without subsequent recurrence, indicates a convulsive tendency, which may be the predisposing factor of epilepsy in the offspring.

(*c*) *Clinical groupings according to the time of the seizures.*

1. Nocturnal and Diurnal Seizures.

Reference has already been made (p. 47) to a type of seizure, which occurs only during sleep. This is an especially interesting variety of epilepsy, as it is a common observation, that in the event of a person, subject to such seizures, falling asleep during the day, a fit is almost certain to ensue.

Attention has also been directed to the great frequency of epileptic fits during sleep, when describing the several types of the disease. The major type of fit is frequently observed only during sleep, and such a condition is illustrated in Chart 3. In some of the combined types, the major seizure occurs during the sleeping, and the minor during the waking hours. Minor seizures, on the other hand, rarely occur only during sleep.

Daily rhythm. The usual incidence of epileptic fits is an irregular periodicity. But there are certain times when their occurrence is more common than others. There exists, moreover, a constancy in the time-incidence of seizures in individual cases. Thus some epileptics have their attacks only during sleep, others at about the time of rising in the morning; more rarely only during the evening hours. One patient invariably had his first nocturnal fit at 10.15 p.m. In others, again, some exciting cause appears to be necessary for their recurrence, such as fatigue, a heavy meal, the excitement of the play or of a dance.

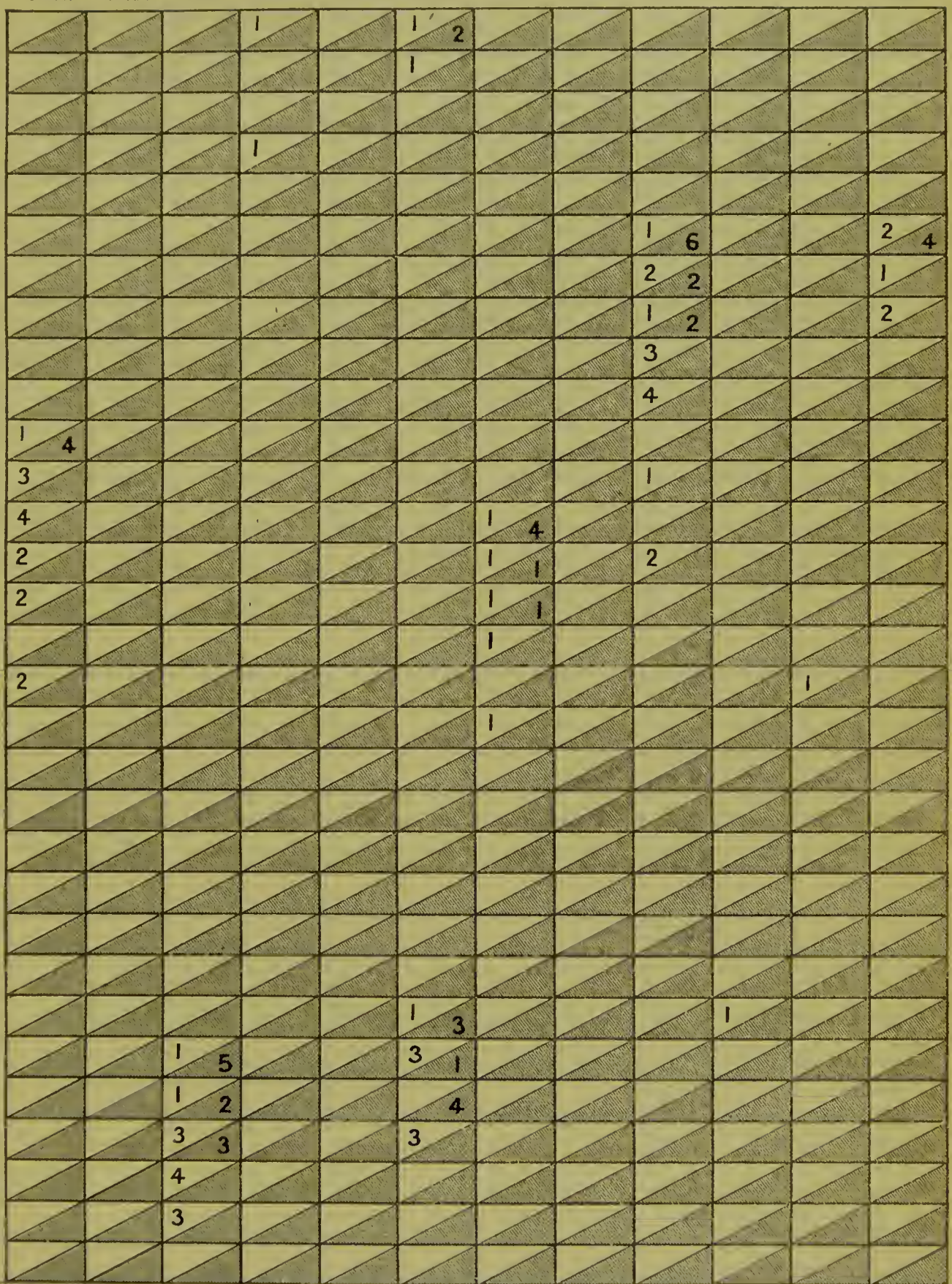
Some writers have endeavoured to account for the incidence of the seizures by the normal variations which take place in the alkalinity of the blood.[1] Haig[2] has explained the more common incidence of epileptic fits during the early morning hours and forenoon, on the ground of the existence at this time of the "alkaline tide," which sets free uric acid and favours its circulation in the blood stream. There is reason to suppose also that fits are more common during the process of digestion than at other times.

Various tabulated summaries are found in works upon epilepsy, giving the hourly incidence of large numbers of

[1] See *Arch. de Neurologie*, 1897, ii. p. 465. [2] Haig, *op. cit.*

HART 7.

An Average Case of Serial Epilepsy.



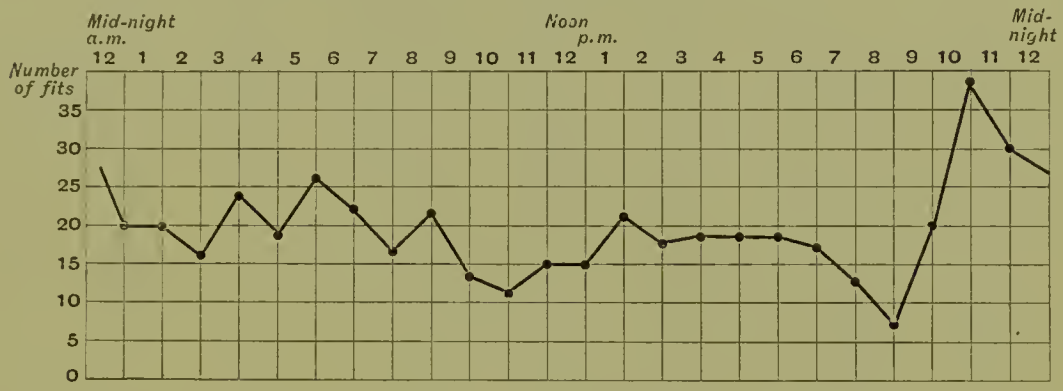
Face page 106.

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fits. Tables of this kind are found in the works of Spratling,[1] Clark,[2] and others. It is seen from these that there is no hour of the day or night, which is free from the occurrence of epileptic attacks, although fits are more common at some hours than at others, the three most frequent periods being 3 to 5 a.m., noon to 2 p.m., and 9 to 10 p.m.; the least common being in the late afternoon, from 5 to 7 p.m.

I have endeavoured to test these points by noting the hourly incidence of fits over some weeks, from a number of cases of the major type of epilepsy at the Chalfont Colony, with the following results:

Chart 11 *showing the hourly incidence of major epileptic fits from sixty-two epileptics at the Chalfont Colony.*



This chart was constructed from the observations made upon sixty-two epileptics, working both in the house and out of doors, over a period of eight weeks. It shows generally:

(*a*) That fits are more common during sleeping than waking hours.

(*b*) That the diurnal fit-incidence remains at a relatively constant and mean level until the evening, reaching its minimum about 8 p.m.

(*c*) That the fit-incidence attains its maximum at about 11 p.m. and the early morning hours, from which time there is a progressive fall until mid-day.

(*d*) The great and sudden rise in the fit-incidence between 10 and 11 p.m. corresponds to the not uncommon onset of fits during the hours of early sleep.

The time-incidence of epileptic fits resolves itself into the following two factors:

1. The incidence in individual cases. Here fits may occur

[1] Spratling, *op. cit.* p. 194. [2] Clark, *Med. News*, 1903, p. 105.

constantly at the same hour, or approximately to the same times. On the other hand, the fit-incidence may be quite irregular, and dependent upon various occasional and accidental causes. The administration of bromides may change the times of the seizures; thus fits occurring during sleep have been transferred to waking hours by aid of treatment.

2. The incidence when studied in bulk. The chart shows that fits may occur at any time, but that there exist certain periods in the twenty-four hours when they are more common: these are during the early hours of sleep and in the early morning;[1] while the hours most free from attacks are during the middle of the day, the afternoon, and the evening.

2. Senile Epilepsy.

This is a term applied to epileptic convulsions, when they occur in later life. The age, after which epilepsy should be regarded as "senile," has been variously stated by different writers; but such a demarcation is purely artificial. There are some authorities who consider this form of epilepsy as identical with idiopathic epilepsy, while others regard it as an epileptoid condition dependent upon arterio-sclerosis.

Reference to the age at onset Table (No. 5) will show that a considerable number of cases of epilepsy commence after forty years of age, and that certain of these cases present a hereditary family history of epilepsy; viz. forty cases, of which four had such a history, or ten per cent. It is therefore clear that as the years progress before the onset of epilepsy, the hereditary influence tends to be less obvious, for the reasons given on an earlier page. It cannot, however, be said that the hereditary influence in late epilepsy is entirely in abeyance: and as the majority of the cases of late epilepsy show evidence of atheromatous, or arterio-sclerotic changes, it is most likely that the latter condition acts as a determining cause in those who are hereditarily predisposed to the disease.

The clinical features of "late epilepsy" may be thus described. It occurs both as the major and the minor type of the disease, but the attacks are less severe and less frequent than those seen in the earlier years of life. The existence of mental failure is less common, and, when it occurs, is less pronounced than in the epilepsies commencing under twenty years of age. Stigmata

[1] Pick, *Wien. Med. Wochensch.*, 1899, nr. 30.

CHART 8.

A Severe Case of Serial Epilepsy, with small Intervening Periods of the Status Epilepticus.

	JAN.	FEB.	MAR.	APRIL	MAY	JUNE	JULY	AUG.	SEP.	OCT.	NOV.	DEC.
1			13 4	1				1		1		2
2		1	6		1						3	1
3						1						
4			3	4		1						
5				1								1 3
6		2										
7			3		7 13							2 1
8			4	2	90 15			1			4	
9			2								4	
10					14						2	4
11		1			10 4		1				1	1
12		2	2 1	2	7 1		1					
13			2		1							
14			3		24	1				4		1 1
15			1		7 8	7		1				1
16			3			6						
17			1		1							
18		9 3	4		1		6 1					
19					5 1		10 3	1				
20		7			3		9 1					
21	1	9 25	4			1	7 1					
22		4	2		2		1		1	2		2
23			3				2			2		
24		4	2		3					9	2	
25		7		1	1				2			
26			14		5 2				1			
27			4		6 2				4	2		1
28			2 2		1		1					2
29		2	2		1		1			3		
30					1	1					2	
31										7		
TOTAL												

face page 108.

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of degeneration are only rarely observed. In character this type of epilepsy may be described as yielding satisfactorily to treatment, the number of cases which become confirmed epileptics being relatively small compared with those which show considerable improvement, or even arrest of the fits (Table 32).

THE ONSET, COURSE, AND TERMINATION OF EPILEPSY.

Onset. Epilepsy commences in a variety of ways. First, by the development of the minor, or "petit mal" type, which may run a course of months or years, before the onset of convulsive seizures. This is a mode of onset accompanied by difficulty and delay in diagnosis, as the significance of the minor attacks may be entirely unrecognised, until a major fit establishes the true nature of the malady.

Secondly, by the development of the "grand mal," or major type, with frequently recurring seizures and the establishment of the confirmed disease.

Thirdly, by the recurrence, at longer or shorter intervals, of attacks during sleep, whose existence may be completely unknown to the patient, and which are only recognised either accidentally, or from the occurrence of a seizure during the day. This is the *nocturnal type* of epilepsy, a variety of the disease which it is possible to conceive may exist throughout life unknown to the sufferer.

Fourthly, by the occurrence of a single fit, months or years before there is any repetition of attacks. It is in connection with this method of onset that an interesting form of commencement of the disease is observed, characterised by diminishing intervals of time between the several seizures, or by an increasing number of attacks during the early years.

The following cases may be cited in illustration:

(1) Fits commenced at fifteen years of age. There were three fits in the first year, four in the second, nine in the third, ten in the fourth, showing a steady increase in the number of attacks as the disease became established.

(2) A patient had his first fit when aet. five years, the second when aet. eleven, the third when nineteen, the fourth when twenty, and the fifth and sixth when twenty-one; an

illustration of diminishing intervals up to the establishment of the malady.

(3) The first fit occurred at twelve years of age, the second after eight years' interval, the third after five, the fourth after five, the fifth after two, and then three fits in succession after three years.

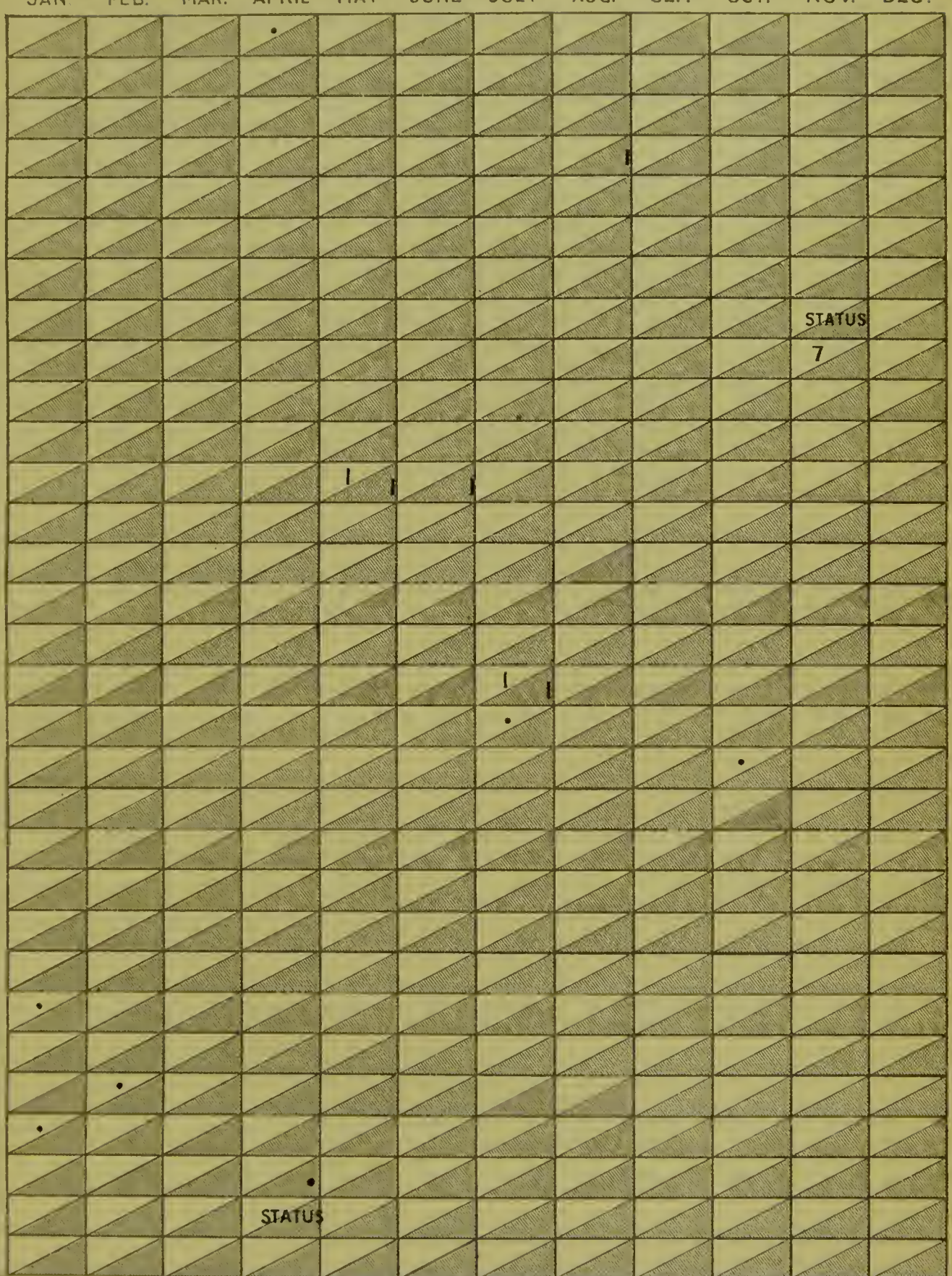
(4) Between the first and second fits—20 months.
„ second and third „ —12 „
„ third and fourth „ —10 „
„ fourth and fifth „ —11 „
„ fifth and sixth „ — 5 „
„ sixth and seventh „ — 3 „

In view of the long interval between the first and second, or even between the second and third attacks, it is difficult to state how soon after the onset the disease may become established. Moreover, it is the existence of such long intervals that occasionally interfere with the successful treatment of the disease in the early years, as it is difficult to persuade a patient to prolonged treatment, when from an absence of further recurrence he is inclined to believe that no further attack will ensue.

Fifthly, epilepsy may arise in an acute form, as in the status epilepticus. This has been seen to occur in some of the so-called puerperal eclampsias, which are in many cases nothing else than serial epilepsy, or the status epilepticus. A similar onset is also observed in childhood, when a convulsive attack, sometimes of marked severity, may attend the onset of infantile hemiplegia. There were also a few instances in which the idiopathic disease in adult life commenced with an attack of status epilepticus.

Course. The disease having commenced in one of the methods just described, and its existence, as a confirmed malady, having been established, it is necessary to enquire as to the general course, which a given case of epilepsy is likely to pursue. The consideration of this subject can only be carried out by the prolonged observation of a large number of cases, in which the incidence of the seizures is carefully and regularly noted. Therefore, facts obtained from cases under treatment in Institutions are likely to be more trustworthy than those obtained from the statements of patients attending the out-patient department of a hospital. I have appended two

CHART 9. ILLUSTRATING SHORT PERIODS OF STATUS EPILEPTICUS.



	JAN.	FEB.	MAR.	APRIL	MAY	JUNE	JULY	AUG.	SEP.	OCT.	NOV.	DEC.
TOTAL	2	1	0	STATUS	2	1	3	1	0	1	STATUS	—

face page 110

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tables, showing the periodic frequency of epileptic attacks—the first based upon the statements of 316 epilepties attending the out-patient department of a special hospital, and the second from the records of 300 epilepties resident in an Institution.

Table 18, *giving the total number and percentage frequency of epileptic attacks from* 316 *cases attending Queen Square Hospital.*

Frequency.	Total Cases.	Percentage.	
Daily fits - -	40	12·6	
Weekly fits - -	96	30·3	73·2
Monthly fits -	96	30·3	
Quarterly fits -	65	20·5	
Yearly fits - -	19	6·0	
Total	316	99·7	

Table 19, *giving the total number and percentage frequency of epileptic attacks from* 300 *cases at the Chalfont Epileptic Colony.*

Frequency.	Total Cases.	Percentage.	
Daily fits - - -	46	15	
Weekly fits - -	133	44	90
Monthly fits - -	94	31	
Quarterly fits - -	12	4	
Yearly fits - -	12	4	
At longer intervals -	3	1	
Total	300	99	

A comparison of these two tables shows that the colony cases provide a higher percentage (90%, as contrasted with 73%) with frequently recurring attacks (daily, weekly, and monthly), and a somewhat smaller percentage which recur at long intervals, such as a year or more.

It is, however, readily understood that the cases of epilepsy, which seek admission to an epileptic colony, are more likely to be severer types of the disease than those who are able to attend to their occupations or household duties, and visit the hospital occasionally for treatment.

The incidence of epileptic fits therefore is seen to be frequent, attacks which may be counted by the week being the type which is commonly found in most instances of the confirmed malady.

It is, however, possible to further subdivide the colony cases, according to the clinical type of the disease, and to show numerically, the relative frequency of the several clinical forms or seizure combinations. This has been done in the following table:

Table 20, *showing by further subdivision of the Colony cases the relative frequency of the several types of fit.*

	Major.	Minor.	Combined.	Serial.	Totals.
Daily - -	10	9	27	0	46
Weekly - -	64	3	55	11	133
Monthly - -	43	0	21	30	94
Quarterly - -	8	0	2	2	12
Yearly - -	11	0	0	1	12
Longer intervals	3	0	0	0	3
Totals	139	12	105	44	300

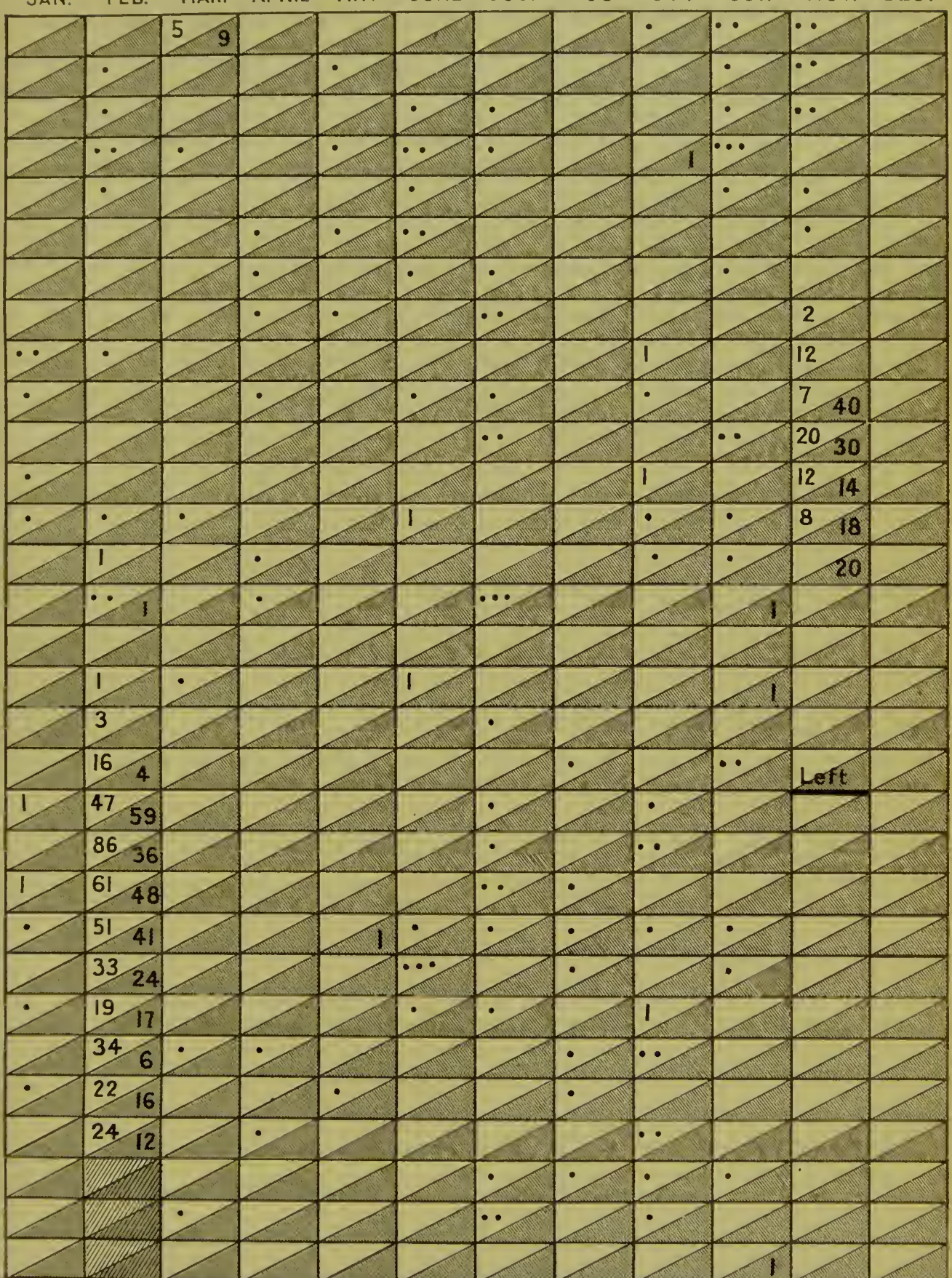
Of the several types, the combined is more frequently of daily occurrence, than the major or minor types. In the major type of the disease the fits are to be counted usually by the week or month, while single attacks, recurring at long intervals, are solely of the major variety. In the minor type, fits are of such frequent occurrence as to be most suitably counted by the day or the week. In the common form of serial epilepsy, where the batch consists of several attacks during one or two days, there is usually a monthly incidence, although in rarer cases they may recur weekly; the appearance of a serial outburst once or twice a year was also noted. In these cases the series is usually more severe, and lasts a greater number of days.

Long Remissions. Epilepsy is a disease characterised by an irregular periodicity in the incidence of the seizures, and accordingly there are seen remissions, sometimes of several years, which are a frequent, if not a characteristic, feature of the disease.

There are three periods in the course of the malady when remissions are prone to occur:

1. Between the first and second fits before the disease becomes satisfactorily established. It may be contended that this is not a true remission, and not therefore comparable to those which occur after the malady becomes pronounced. It is, however,

HART 10. ILLUSTRATING PROLONGED PERIODS OF STATUS EPILEPTICUS.



Face page 112

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an important remission, the recognition of which may prove of value in the subsequent treatment of the disease.

Of my series of 1000 cases, the following showed periods of varying duration, from one to ten years, between the first and the second fits:

1 year's interval in	8 cases.	7 years' interval in	4 cases.
2 years' „	6 „	8 „	2 „
3 „	2 „	9 „	1 „
4 „	3 „	10 „	1 „
6 „	3 „		

An interval of thirty years was noted in one case, the first attack occurring at ten years of age and the second at forty; but this patient was of alcoholic habits, and has not been included in the above table.

2. The second period of remission is found during the years of childhood, antecedent to puberty, in those whose fits commenced in infancy. This is one of the most remarkable features of the disease, as during this period the processes of brain growth and development are usually active. Nevertheless, it corresponds to a period during which the onset of epilepsy is less common, as is readily seen from a study of age at onset table (p. 19). The common epoch for this remission is from five to twelve years of age, but it may also take place between other age-periods, as is shown in the following table:

5	cases showed	remission	between	5 and 12 years of age.
3	„	„		7 and 17 „
3	„	„		7 and 14 „
2	„	„		3 and 9 „
2	„	„		7 and 10 „
2	„	„		10 and 16 „
2	„	„		9 and 15 „
1	„	„		7 and 13 „
2	„	„		3 and 13 „
1	„	„		6 and 15 „
1	„	„		8 and 15 „
1	„	„		10 and 14 „
1	„	„		7 and 19 „

With the onset of puberty the commonest cause of epileptic attacks is introduced, hence the relapse which takes place at or about this epoch in those cases is explained. It may be urged

that the fits which were present before the remission were not genuinely epileptic in character; but the statement is advanced that the presence of this remission, and the subsequent relapse, stamps the malady as essentially epileptic in character.

A point of unusual interest, which does not seem to have received previous attention, is the occasional existence of a remission extending over the ages of puberty and early adolescence, in those who had previously suffered from fits, and who eventually became confirmed epileptics. Nine such cases were observed in the series under consideration. The remission corresponded to the following age periods:

Remission from	12	to 22	years of age.
„	12	to 28	„
„	14	to 18	„
„	14	to 29	„
„	14	to 33	„
„	14	to 36	„
„	15	to 27	„
„	16	to 21	„
„	18	to 21	„

It is during the above-mentioned age-periods that the onset of epilepsy is most common, the limits corresponding to the periods of puberty and adolescence. Why in some cases this time should correspond to a spell of complete freedom from fits is not apparent: but it is merely a further illustration of the extraordinary uncertainty of the disease, and of the way in which remissions may occur without any obvious explanation.

3. Remissions occur after the complete establishment of the disease, being induced either by treatment, or arising spontaneously. An examination of the series of 1000 cases revealed many instances, in which remissions persisted for a number of years, but which were succeeded by a return of the characteristic attacks. Many cases of "arrested" epilepsy are no doubt instances of long remission, but fuller reference will be made to this subject when the arrest or cure of epilepsy is under consideration. Two points especially call for notice in this connection: on the one hand, long remissions may occur under suitable treatment, to be followed by relapses when medication is omitted: and, on the other hand, a remission may be broken by accidental circumstances, such as a blow upon the head, a fall, child-birth, or an acute inflammatory disorder.

In the following table is given the number of cases with remissions and their duration:

A remission of	2 years in	5	cases.
,,	3 ,,	16	,,
,,	4 ,,	7	,,
,,	5 ,,	4	,,
,,	6 ,,	1	,,
,,	7 ,,	2	,,
,,	8 ,,	1	,,
,,	9 ,,	1	,,
,,	10 ,,	1	,,
,,	12 ,,	1	,,
,,	13 ,,	2	,,
,,	15 ,,	1	,,
,,	19 ,,	1	,,
,,	30 ,,	1	,,
Total, -	-	44	cases.

Termination. Epilepsy is a progressive degenerative disorder, ending either in a cure in a small percentage of cases (stated approximately at about 10 per cent.), or in mental infirmity, delusional insanity, and dementia. The percentage of epileptics who retain their mental integrity, or pass into the several degrees of dementia, has been variously stated, but this subject will be discussed in the next chapter.

Out of 150 confirmed epileptics at the Chalfont Colony in whom the disease began about the usual time of puberty, and who were under constant observation for periods varying from two to ten years, I found the following percentages as showing the modes of termination of the disease:

1. Discharged "cured,"	4 cases,	2·6	per cent.
2. Retention of a normal mental state,	8 ,,	5·3	,,
3. First degree of dementia,	35 ,,	23·3	,,
4. Second ,, ,,	40 ,,	26·6	,,
5. Third ,, ,,	41 ,,	27·3	,,
6. Delusional insanity or other permanent states requiring asylum care,	20 ,,	13·3	,,
7. Died as a result of a fit or fits,	2 ,,	1·3	,,
Total, - - -	150 cases,	99·7	per cent.

It is therefore obvious that, amongst those epileptics, in whom the disease is so pronounced that they are unable

to earn their living by the ordinary methods, and have in consequence to be cared for in an Institution, the greater proportion—viz., 77 per cent.—exhibit mental impairment in a greater or less degree, and 13 per cent. pass into a state of delusional insanity, requiring care and attention in an asylum.

The *causes* and *manner of death* in epilepsy have been described by Spratling from a study of 150 cases under observation at the Craig Colony. From this it is seen that an appreciable percentage of epileptics die as a direct consequence of a fit, such as may occur from rolling over upon the face and asphyxia. One of the causes of sudden death is stated by Brouardel to be an attack of epilepsy, the fatal result being due usually to the entrance of food into the air passages. Death in epilepsy may also be due to asphyxiation resulting from the position assumed by the patient during or after the fit. Other causes are found in accidents resulting from a fit, such as a fall, whereby the head is seriously injured, or a part of the body is burnt, or from drowning, or from fracture of the spine. Whether death may result from paralysis of the respiratory centre, without the intermediation of a fit, is a point upon which there is doubt, although some authors have expressed their belief in it.

Spratling's figures, showing the causes of death in 150 cases, have been appended in the subjoined table:

Table 21 *showing the causes of death in* 150 *cases of idiopathic epilepsy.*

1.	Suddenly as a result of a fit,	5 per cent.
2.	From status epilepticus,	23 „
3.	From accidents during a fit,	12 „
4.	From pulmonary, chiefly tuberculous, disease,	24 „
5.	From organic heart disease,	10 „
6.	From all other causes,	26 „

From the above it is seen that rather less than half the number of epileptics die from some cause directly attributable to their disease Habermaas'[1] figures give a somewhat higher percentage—viz., 60 per cent.—as dying as a result of epilepsy itself; while the figures given by Worcester indicate that about 25 per cent. succumb directly to the effects of the malady.

[1] Habermaas' figures are the following: Epilepsy is curable in 10·3 per cent.; 17·3 per cent. of epileptics remain free from mental disturbances; 60 per cent. die as a direct result of epilepsy. The duration of life is approximately 25 years.

The *mean age at death* is variously stated. Spratling gives 29·4 years, and Snell, 33 years. At the Massachusetts Epileptic Institution it was found to be 39 years,[1] and in the London County Asylums, 40 years (Lord).[2] Muirhead from the records of the Scottish Widows' Insurance Association puts it at 48 years.[3] The great divergence between the maximum and minimum ages is no doubt due to the different type of epilepsy studied by the several observers.

[1] *Boston Med. and Surg. Journal*, 1905.

[2] *Jour. of Mental Science*, July, 1899.

[3] Scottish Widows' Fund. Report of Medical Officer, 1902.

CHAPTER VI.

THE MENTAL STATES FOUND IN EPILEPSY.

Epileptic temperament—Pre- and post-paroxysmal psychoses—Acute epileptic dementia—Acute epileptic mania—Transitory delusional states—Automatism—Psychical epileptic equivalents—Psychical epilepsy—Epileptic ambulatory automatism—Epileptic mania—Impulsions—Dreamy mental states—Stupor—Delusional states—Miscellaneous equivalent states—Substitution phenomena—Epileptic dementia—Dementia an integral part of the disease.

In the account of the epileptic phenomena, thus far recorded, motor and sensory disturbances of a paroxysmal character have been mainly described. The present chapter will be devoted to an account of the psychical phenomena, which are seen in the course of this disease, and which are none the less a part of the "symptom-complex," although they are less frequent in their occurrence, more bizarre in their manifestations, and comparable in many ways to similar phenomena of a non-epileptic kind.

The mental states found in epilepsy may be classified and described, according as they are observed:

1. In the epileptic character and temperament.

2. In the paroxysmal psychoses, which either precede or succeed the convulsive phenomena.

3. In the paroxysmal psychoses which replace single fits, or series of fits, and which have been regarded as psychical equivalents of the convulsive seizures.

4. In the permanent interparoxysmal mental condition, which is observed in most epilepties, and which should be looked upon as a well-marked and characteristic feature of the confirmed disease.

It is stated by writers upon epilepsy, that the convulsions of the disease necessarily impair the integrity of the mental

faculties. This, however, is not always the case, for statistical evidence will be brought forward to show that some epileptics retain, under favourable conditions and for prolonged periods, quite a marked degree of mental acuteness. Many epileptic seizures, on the other hand, produce some temporary mental confusion and interference with memory, or lead to a psychical state characterised by the performance of automatic actions. Such conditions are transient and may be repeated from time to time at long intervals, without any obvious permanent impairment or blunting of the mental faculties.

1. THE EPILEPTIC TEMPERAMENT.

It is rare to find epileptics who do not present some form of mental obliquity, which may be only slight and unobtrusive, but the possession of which is a feature of their hereditarily degenerative disposition.

The memory is usually impaired for recent events; or it may be phenomenally good and exact, but of a perverted kind, so that events and details, of little or no practical use, may be recalled with an accuracy and promptness which astonishes the hearer.

Epileptics are on the whole self-opinionated and egotistical, and possess a conceit and assurance which is out of all proportion to their achievements; their conversation also is usually prolix and pretentious. In character they are mobile and unstable, rising and falling to extremes of gaiety and despondency. Their mental perspective is in consequence blurred and disproportioned, giving to their views and methods an importance which cannot be legitimately claimed for them. On the other hand, there have been epileptics who were conspicuous for the tenacity, even amounting to obstinacy, of their likes and dislikes; and some celebrated men, who were known to be, or were suspected of being, epileptics, were distinguished as much by their tenacity of purpose as by the greatness of their ideas (Féré).

The majority of epileptics possess a religious fervour which forms a marked feature of their disease, and contrasts strongly with their actions, which are often perverted, passionate and immoral. Their ideas of right and wrong are often vague.

There are many instances in history of great religious movements, which are said to have been initiated under the influence

of epileptic ecstasy and ideas. Moreau has stated his belief that Mahomet was an epileptic, and that the fanaticism, characteristic of his actions, arose from the maniacal delirium associated with epileptic seizures. It is probable that Swedenborg was subject to epileptic attacks, and it is known that Ann Lee, the founder of the Shaker community, was a confirmed epileptic[1]; and there are many other instances in history of great leaders, who were believed to have been subject to epileptic fits.

The extremes of the epileptic character are no doubt psychical parallels to the convulsive paroxysms, which form the leading feature of the malady. Hence we find moods of pugnacity and hastiness, amounting at times to violence, alternating with periods of laziness and lethargy, which make the confirmed epileptic difficult to live with, and often useless as a worker.

Although there are striking exceptions their want of initiative is characteristic; and mainly on this account the majority require supervision and direction.

Many epileptics, moreover, show habitual irritability of temper and awkwardness of disposition, while others are imbecile, demented, delusional, or liable to dangerous impulses.

Their judgment is feeble: they are frequently credulous and mystical, and given to superstitious ideas and fancies.

In addition to these qualities and defects, epileptics may show abnormalities both of emotion and of will power. Under this heading the psychasthenic states, grouped under the obsessions, may be mentioned. Objectively these are characterised by indecision and doubt, by an exaggerated self-consciousness, by apprehensions and fears, by feebleness of will power and hesitancy, which prevent resistance to certain impulsions, to decisive action, and to the accomplishment of ordinary every-day actions. (Ballet.)

None of these last features should be regarded as in any way peculiar to epileptics, for they are the common attributes of most forms of mental degeneracy and deficiency.

2. THE PAROXYSMAL PSYCHOSES—"EPILEPTIC INSANITY."

The paroxysmal mental states in epilepsy may be described according as they are found: first, incidental to the convulsive

[1] Skae, *Journ. of Mental Science*, 1874.

seizures, either as prodromal phenomena, or as immediate sequelæ: and, secondly, as outbursts of acute mental disorder in the interparoxysmal periods, when they are believed by some writers to replace the convulsive fits. As the latter are, on this hypothesis, held to be psychical equivalents of the motor paroxysms, they will more suitably be described under that designation (p. 126).

Under the term paroxysmal psychoses, therefore, are included all those hallucinatory, delusional, maniacal, melancholic, and psychasthenic states which temporarily precede, succeed, or replace, more especially the major convulsions, and which may be regarded as constituting Epileptic Insanity. The acute psychoses of epileptics do not differ essentially from similar conditions occurring apart from epileptic attacks, except perhaps in the precipitancy of their onset, the intensity of their symptoms, their shorter duration, and the greater abruptness of their termination.

A. Pre-Paroxysmal Psychoses.

These ought to be regarded as in reality prolonged or modified phases of the stage prodromal to the convulsions, already described. The prodromata are commonly of a psychical character, and consist chiefly of fleeting irritability, feelings of apprehension, of fear, and of a dream-state, inhibition of thought and lethargy, increased impairment of memory, and sometimes definite hallucinations, and even a delusional phase. A form of pre-paroxysmal psychosis of great interest, but less frequently seen than those just mentioned, is the feeling of good spirits and of exceptional well-being, which precedes the onset of an attack in some cases. These phenomena not infrequently precede for a day or two the onset of a fit, and, in cases of serial epilepsy, the prodromal mental symptoms may be accompanied by an increase in the frequency of the minor seizures, which herald the approach of the serial outburst.

Another variety of pre-paroxysmal psychosis is seen in the prolongation of the psychical aura, which has been already described. This is not infrequently accompanied by symptoms of an obviously psychical type, such as excitement bordering almost upon mania, or of intense melancholy, rarely, however, of suicidal character; while the subjective sensations

are of an obsessional kind, such as a fear of something awful about to happen, of terrible thoughts, or of a feeling of unreality. There have also been described impulses more or less sudden, the patient doing strange things and then falling down in a fit.

The pre-paroxysmal psychoses are well worthy of careful recognition, as those who live with, or have to attend to, epileptics are often by these means able to predict the near approach of a seizure.

B. Post-Paroxysmal Psychoses.

The immediate sequelæ of a convulsive seizure, or series of seizures are found, as we have seen, in motor, sensory and reflex disturbances of a temporary character.

In some instances, however, the phenomena of the after-stage do not resolve in the accustomed manner, but are, on the one hand, prolonged for variable periods, or on the other hand, assume phases which are unusual, or exhibit exaggerated forms of action, which are characteristic of acute maniacal outbursts. These temporary, though sometimes prolonged, effects are part of, and are inseparable from, the convulsions, disappearing when the effects of the attack have passed away, but constituting in some cases the clinical type of the disease.

The post-paroxysmal psychical phenomena are represented by the following temporary conditions:

1. Acute epileptic dementia.
2. Acute epileptic mania.
3. Transitory delusional states.
4. Post-paroxysmal automatism.

1. *Acute epileptic dementia.* This condition is seen most commonly after a severe convulsive seizure, a series of seizures, or the status epilepticus, and appears to be nothing else than a prolongation of the stuporous after-stage of the major epileptic convulsion. It may last a few days, or even a week or two. The patient lies in a state of profound physical and mental lethargy, from which he may be temporarily aroused to answer questions and to be given nourishment. In some cases reflex action is lost, or diminished—the pupillary light reaction being slow, and the knee-jerks abolished or defective; in others the pupil reflexes may be normal and the knee-jerks exaggerated. Myoclonic jerkings or "jumps" of the limbs, comparable to

what have been described as occurring in the pre- and inter-paroxysmal periods in many epileptics, are phenomena sometimes seen during the stuporous period, and would appear to represent the ghosts of the previous convulsions, in the form of minor seizures.

This state of stupor gives place to one in which the mental faculties begin to reappear, at first slowly. With this re-appearance, delusions, hallucinations, and a restless, confused, and dazed condition of mind are not uncommonly found. These symptoms gradually pass away, and the patient assumes the mental attitude which was present before the series of convulsions.

Repeated attacks of this kind, however, exert in course of time a markedly deteriorating influence upon the general mental state. A perusal of Table 28, page 150, will show that a large percentage of the cases of serial epilepsy develop eventually into epileptic dements of the confirmed type.

An illustration of acute epileptic dementia may be taken from the case of a confirmed epileptic, who was subject to repeated attacks of status epilepticus. The onset of the series was heralded by some increase in the frequency of the seizures, coincidental with various prodromal symptoms of an unusual kind. These consisted of a phenomenal increase of the appetite amounting to voraciousness, a muddy complexion, constipation, a furred and dirty tongue, and an unpleasant odour of the cutaneous secretions. Following upon the preliminary increase in the number of the attacks, seizures began to succeed each other in rapid succession, and the temperature rose to 101° F. This continued for about forty-eight hours, when the attacks gradually subsided and the convulsionless comatose stage was reached. During this stage the patient could be roused temporarily to be given nourishment. The breathing was of the Cheyne-Stokes character, the knee-jerks were not obtained. Three days later he became less stuporous, and his mental state was that of delusional confusion; he talked to people whom he believed to be about him and whom he thought were saying ill of him. About this time he had lost all sense of locality and was unable to call by name those whom he was accustomed to see around him. After a week this stage gave place to one of great physical debility, during which he was unable to do any work; and in three weeks he had returned

to his natural mental condition, which may be described as one o suspiciousness, quarrelsomeness, and irritability.

This phase of acute epileptic dementia is a highly characteristic phenomenon amongst epileptics under observation in institutions. It is a condition demanding great care and attention, as the patient requires skilled nursing, having to be fed during the earlier stages of stupor, and watched and attended to during the later delusional periods. It is sometimes associated with symptoms of a cataleptic nature, and anaesthesia both of the skin and mucous membranes (Binswanger). It is a condition from which recovery ought to take place, under favourable circumstances.

A form of mental clouding, consisting mainly of a temporary dulling of memory and of the finer mental processes, is sometimes observed during a transient increase in the frequency of the seizures, more particularly at the commencement of the disease. This mental blurring should not be taken for other than a temporary epileptic dementia, as, in the majority of cases in which it occurs, it clears up under the judicious administration of the bromides, or when the patient is removed from unsatisfactory domestic or hygienic environment.

2. *Acute epileptic mania.* This is a rarer post-paroxysmal sequel than the preceding, but on account of the intensity of the psycho-motor disturbance is more difficult to manage. The attacks of mania usually follow the major seizures, and are characterised by extreme frenzy and maniacal excitement. These patients rush about blindly assaulting and striking, and require to be controlled by a number of attendants, or even placed in the padded room; consciousness is entirely obliterated during the attacks, so that the patient has no knowledge of what has happened. The attacks of mania are usually brief, but may be prolonged for twelve or more hours. One patient, a strongly-built ex-policeman, who was subject to frequent minor seizures, had a major fit about once or twice a year, and these attacks were invariably followed by maniacal outbursts of extraordinary violence, lasting several hours.

There are, however, many degrees of post-paroxysmal excitement, from mere talkativeness, tiresomeness, and exhilaration of spirits, up to the most pronounced frenzy which it is possible to conceive.

It is also noticeable that the attacks of excitement do not

necessarily immediately succeed a fit, an interval of a day or two sometimes elapsing between the convulsion and the onset of the mental symptoms.

The phenomena of excitement and mania are more common in cases in which the fits are far apart, recurring, as in one of my cases, about once a year; they are also more frequent in the alcoholic and traumatic cases than in those of ordinary idiopathic epilepsy.

Epileptic mania is an important psychical equivalent between seizures, when it constitutes the acute epileptic frenzy designated by Falret "grand mal intellectuel," and will be again referred to on page 133.

3. *Transitory delusional states.* It has been already mentioned, when describing post-paroxysmal dementia, that a phase of delusional confusion usually intervenes between the temporary acute dementia and the restitution of the normal mental attitude.

But there are other cases in which delusional states may occur after single, or serial, fits, in which no pronounced dementia has been present. In these cases the patients develop delusions of a simple character: they imagine that their food is poisoned, that their attendants are thinking or speaking evil of them, etc. This condition usually lasts for a few days, during which the epileptic requires to be fed and attended to; but it passes away, and the normal mental state is regained.

On the other hand, a number of epilepties pass eventually into a state of continued delusional insanity, requiring asylum treatment, a condition which scarcely demands consideration here, as it does not differ from that occurring in those who are not subject to epileptic fits. My statistics show that 13·3 per cent. of the cases of confirmed epilepsy terminate in this way.

A minor form of transitory mental disturbance, not amounting to acute dementia, and unaccompanied by delusions, was observed, sometimes after short series of fits. It was characterised by a state of mental facility, fatuousness, and "silliness," in which the patients laughed readily at minor matters, and were unable to apply themselves to work. It succeeded incomplete major seizures, and lasted from two to three days, but cleared away entirely and left the patients in a fit state to resume their usual avocations.

4. *Automatism.* Most epileptic seizures are followed by some

kind of automatic action, which may vary from simple movements of unbuttoning the clothes up to the most pronounced violence requiring restraint. Probably the commonest post-paroxysmal automatic action is that of undressing, to which exposure of the genital organs is closely related. Of others which were observed, may be mentioned: crouching under the table and "uttering sounds like an animal," removing the contents of the pockets, fumbling with the clothes, and running away; one patient invariably removed her artificial teeth.

The subject of epileptic automatism is one of great interest and importance, as it forms a possible and likely explanation of many actions, involving medico-legal considerations. As just mentioned, it is a characteristic phenomenon of the after-stage of some epileptic fits; or it may replace the fit, when it is recognised as psychical epilepsy, or as a psychical epileptic equivalent, under which circumstances it may be observed in various bizarre and criminal actions, more particularly with reference to sexual perversions, theft, and incendiarism, which are described more fully on a subsequent page.

It is hardly within the province of this work to enter into the *medico-legal* aspects of epileptic automatism, whether found in the form of simple wandering, unusual actions, or indecent behaviour: but a word may be said as to the responsibility of the person concerned. It may be difficult to decide whether the acts, above-mentioned, are done in a wholly, or partially subconscious state; but if it can be shown that the patient is, or has been, subject to epileptic seizures, there is always a reasonable ground for the belief, that an unnatural, unusual or perverted action, may have been effected in a state of post-paroxysmal subconsciousness, or during a psychical phase 'equivalent to' an epileptic attack. The persons subject to such actions, usually present other symptoms denoting their epileptic character, such as, headache, depression, and blurring of the memory, often for remote as well as for more recent events.

3. PSYCHICAL EPILEPTIC EQUIVALENTS.

Most recent writers upon epilepsy agree with the doctrine, that there exists a clinical variety of the disease, characterised by an absence of the usual motor-convulsive, or sensory, phenomena, which are generally held to constitute the epileptic

seizure. This type has received the name of "Psychical Epilepsy," and is regarded as epileptic from the fact that consciousness is disturbed, and memory temporarily in abeyance; although the essential disturbance of consciousness and the objective phenomena of the attack, differ from those which usually occur in epileptic fits. It has, in consequence, been described as a "psychical equivalent"[1] of the motor convulsion, in that it is a psychical state, which is believed to take the place of an epileptic seizure.

In discussing the psychical equivalents of epilepsy, we enter upon a much debated and extremely complex subject, but of enormous importance from both the clinical and the medico-legal standpoint. We have in the first instance to decide, whether the psychical conditions about to be described are in reality equivalents of epileptic seizures: or whether they ought to be regarded as sequelæ of attacks, which are either so slight, or so transient, as to have escaped observation. A further element of difficulty lies in the fact that many of the psychical states, defined as "epileptic equivalents," are not necessarily co-existent with epileptic convulsions, but are found in various forms of psychopathy and of mental deficiency, and in alcoholics.

Although these mental conditions were known to the older writers (Pinel, Esquirol, and others), their significance was first pointed out by Morel; and although their real nature has since been corroborated by many extended observations, there are still writers who believe that the mental phenomena known as "masked epilepsy," are preceded by some form of incomplete epileptic attack.

Within recent years, however, the range of epileptic phenomena has been largely extended, and there are authorities who regard as epileptic, psychical phenomena which are seen in their most protean manifestations, not in genuine epilepsy alone, but as symptoms common to it and the degenerative psychoses.

Viewed from this standpoint, the writings of Lombroso, Aschaffenburg,[2] and others, although materially widening the horizon of the epileptic phenomena, have of necessity removed from the diagnosis the importance of convulsion, or spasm,

[1] This term seems to have been first employed by Hoffmann in 1862.

[2] Aschaffenburg: *op. cit.*; and ref. in *Rev. of Neurology*, 1906.

as the primary feature of this disease. Some writers are inclined to regard "periodicity" as the phenomenon, upon which the diagnosis of epilepsy should be based. Aschaffenburg indeed has endeavoured to show that convulsion, vertigo and emotional depression are not cardinal symptoms of epilepsy. He is rather of opinion that periodic fluctuations of the "psychical equilibrium," which may or may not be accompanied by convulsion and disturbances of consciousness, are the essential features. In his list of the epileptic phenomena, convulsions hold a relatively low position, forming only 42 per cent. of the cases, while 70 per cent. showed variations of the emotional tone, such as periodic depression and excitement, as "epileptic equivalents."

It is well known that psychical periodicity is not pathognomonic of epilepsy, as all the psychopathies present irregular fluctuations in their clinical appearances. Hence the periodic exhibition of many psychical states, described by the criminologists as epileptic equivalents, viz., subjective feelings of fear, of impending disaster, of a desire to wander, to commit suicide or homicide, to steal, to set on fire, various sexual perversions, etc., are, when occurring independently of fits, more cognate to moral delinquency and mental deficiency than to epilepsy proper.

Some of the symptoms above mentioned are no doubt found as psychical equivalents in cases of idiopathic epilepsy, and reference will presently be made to their relation to epileptic attacks; but before doing so, it is necessary to point out that a mental (or psychical) attack should conform to certain conditions, before it ought to be regarded as a psychical epileptic equivalent. These are:

1. Its association in the same person with evidence of classical epileptic phenomena—major or minor fits, or vertiginous attacks with interference with consciousness.

2. A certain uniformity in the type of the attack, recurring paroxysms reproducing the features of former attacks in the same person.

3. A definite relation to the existing epileptic seizures: or its occasional independent occurrence in place of fits, more especially at the catamenial epochs in women.

A feature peculiar to the psychical equivalents is found in their tendency to increase, both in severity and frequency, when the classical seizures lessen or disappear.

They are often preceded by prodromal mental symptoms, consisting of irritability, suspicions, and apprehensions, and may be accompanied by various somatic sensations, corresponding to those which precede the motor convulsions, such as headache, dyspepsia and sensory warnings.

As the name implies, psychical equivalents are phenomena which are believed to replace epileptic convulsive seizures. Mental states similar to them, however, may also precede and succeed epileptic fits, and have already been described as the pre- and post-paroxysmal psychoses.

It may indeed be categorically stated that there is no psychical equivalent condition, which is not also seen as a pre- or post-convulsive psychical phenomenon. I have not infrequently observed in an individual similar mental conditions, occurring both as post-convulsive and as equivalent states.

Illustrations of this may be seen in the following examples: (*a*) a young epileptic, whose post-paroxysmal states consisted mainly of irritability, quarrelsomeness and pugnacity, had occasional transitory phases of a similar character without any antecedent convulsion; (*b*) another presented as pre-paroxysmal features, the symptoms of the psychasthenic dream state described on a later page. Attacks of the dream-state were also observed lasting for two or three days, unassociated with any obvious convulsion, in the inter-paroxysmal periods; (*c*) a third epileptic, whose post-convulsive symptoms were mainly of the nature of cataleptic rigidity and dementia, from time to time showed similar cataleptic stupor states, unassociated with convulsion.

These observations might be used as arguments in favour of the view that the so-called "psychical equivalents" are really post-paroxysmal psychoses, occurring after attacks so slight, or so transitory, as to pass unobserved. It seems to me, however, to be more in harmony with clinical observation and experience, to define as "*psychical epileptic equivalents*" *the mental phenomena of the pre- and post-convulsive states, when they occur without convulsion or spasm.*

In view of this definition there will only be described as equivalents, those psychical states which have their counterpart in the pre- and post-paroxysmal mental phenomena.

The following conditions therefore are alone considered as **psychical epileptic equivalents**:

1. Psychical epilepsy, consisting of two varieties:
 (*a*) Short psychical attacks, or true psychical epilepsy.
 (*b*) Prolonged attacks, or epileptic ambulatory automatism.
2. Epileptic mania, including the impulsions.
3. Dream-states.
4. Temporary delusional states.
5. Catatonic stuporous conditions.
6. Cephalic and "aura" sensations.
7. Miscellaneous phenomena.

1. **Psychical epilepsy.** It is necessary in the first place to refer to this special variety of epileptic attack and to mention its leading clinical characteristics. It may be primarily subdivided into:

(*a*) Short psychical attacks, which are either purely psychical, without antecedent spasm or convulsion, or psychomotor, in which the psychical period succeeds to a temporary and transient motor spasm. This latter type is analogous to, and indistinguishable from, the ordinary forms of minor epilepsy with automatic actions already described (p. 84).

The purely psychical attack of short duration is probably commoner than is generally supposed; it may, or may not, occur alone; in the latter event, short series of psychical seizures are often the prelude of a severe convulsive attack. On the other hand, the convulsive fit may precede the short series of psychical attacks, which seem to follow in the wake of the major cerebral disturbance. Their occurrence in series is one of their chief clinical characteristics; while the coexistence of psychical epilepsy with major fits forms one of the varieties of the combined group of epilepsies already mentioned (chart 6).

The attacks of psychical epilepsy, here described, consist mainly in the performance of simple automatic actions; for instance, a woman who was daily employed in the laundry, during her attacks carried out the movements she was accustomed to do when at work, such as folding the clothes preparatory to placing them in the basket; another patient would rub his hands together, fumble with his clothes, take out his watch, and exclaim, "Ha, Ha!" and then resume his occupation. In other instances, the attacks are of a more complicated nature, resembling the ambulatory form to be

immediately described. These patients will rise up from table and walk away into an adjoining room, where they will perform some action entirely different to what they were doing immediately prior to the attaek.

Spratling[1] also describes cases of psychieal epilepsy, in which the movements during the seizure are repetitions of that work, which the patient is in the habit of doing. Thus one epileptic, whose duty it was to address and tie up pamphlets in the office, during his psychical attack proceeded to tie up papers into bundles; another who was addressing letters when the seizure eame on, repeated the same name and address six times; and a third would proceed to get his broom and sweep the floor when the attack came on.

During the attacks the face is usually pale, and has a fixed staring expression; the pupils are dilated and may not react to light. There is no subsequent recollection of what has occurred during the seizure; although in some eases there would appear to be a state of subconsciousness, in which the patient may hear vaguely what is said, but is unable to reply to questions.

These attacks are characterised by sudden onset, and termination, and usually occur without warning. They are of short duration, and recovery is rapid. In how far they should be regarded as forming a type of seizure distinct from ordinary minor attacks, is a point upon which difference of opinion may be expressed; but from the fact that there is no obvious antecedent motor spasm, and that the interference with consciousness is not such as to forbid the execution of semi-purposive, though unintentional actions, and from their close resemblance to the prolonged psychical seizures next to be described, they have been defined as epileptic equivalents of a purely psychical order.

(*b*) Prolonged psychical attaeks.—*Epileptic ambulatory automatism.* These attacks may last from one or two hours up to several days, during which the patients may travel long distances, and awake to find themselves in some unknown locality. They are usually ushered in by prodromal symptoms similar to those preceding ordinary epileptic seizures, and terminate in a stage of confusion, headache, and exhaustion.

The same kind of alteration of eonsciousness would appear

[1] *Op. cit.* p. 153.

to underly both the short and the long varieties of psychical seizures; being in the former a transient, and in the latter a more prolonged condition. Motor disturbances indicating a convulsive "discharge" from cortical motor areas are absent; but motor phenomena in the form of complicated actions of a semi-purposive, or automatic, order are present. The condition more closely resembles the somnambulistic state than any other, so much so indeed that some states of somnambulism in adults appear to be epileptic in character. The attacks of "automatic wandering" usually occur only at rare intervals, and are not necessarily associated with ordinary convulsive epileptic seizures, although the family history is generally strongly psychopathic, and somatic and mental stigmata of degeneration are present.

"Wandering" is probably more frequent amongst epileptics than is commonly suspected, for it is no rare occurrence to observe this symptom amongst the inmates of an institution for epileptics.

But ambulatory automatism is not confined to epileptics. It is a familiar symptom of chronic alcoholism, dementia, and congenital mental deficiency, and it has also been observed in the hysterical.

Perhaps the features of ambulatory automatism may be best described by reference to an illustrative case, under the author's personal observation.

A married man, aet. 40, after some prodromal irritability and a few days' severe headache, lost all knowledge of his surroundings for a period of ten days, during which time he carried on some of his ordinary duties, called upon his brother on one occasion, and then disappeared from his home in London. He had no recollection of anything until he came to himself again in Hull, where he applied to the police for assistance. During this period of time he had been seen by his wife, his brother, and a friend, none of whom noticed any difference from his usual state, except that he seemed quieter and had a dazed appearance. He had no knowledge as to how he journeyed from London to Hull, a distance of 150 miles, or where he spent the two nights after his disappearance from home. He had disposed of some of his money, but his watch and other contents of his pocket were untouched. The attack was followed by confusion for a few days, insomnia, and severe

headache, but these symptoms passed away and he returned to his normal condition. He never had an epileptic fit, but suffered for many years from paroxysmal headaches. His daughter had chorea, and his elder brother on one occasion had an epileptic convulsive seizure, and on another an acute attack of temporary delusional insanity, requiring restraint.

In this case there existed all the clinical features characteristic of the type of malady under discussion: the neuropathic family predisposition, the personal history of paroxysmal headaches, the prodromal stage of headache and irritability, the prolonged abolition of normal consciousness with automatic wandering, followed by the after-stage of confusion, exhaustion, and headache.

It is not usual to find violence and assault performed during the periods of automatic wandering, but there were two cases in which this was noted; in both the assault was of a homicidal character; but neither patient was a genuine epileptic, in that there was no personal history of the classical epileptic fits, although the family history of one, an alcoholic, was strongly psychopathic; and in the other, a somnambulist, there were obvious structural stigmata of degeneration.

The duration of the wandering periods varies greatly. In the case recorded above the loss of memory lasted for ten days. In Charcot's case the lapse was of eight days' duration, and Colman[1] has recorded three cases, in which the period of attack was twelve hours, two and a half days, and five and a half days respectively. Spratling describes a case in which the attack persisted for twenty-eight days, during which the patient travelled many miles, and carried on business transactions.[2]

2. **Epileptic mania.** "Grand mal intelleetuel" of Falret. There has already been described, as a post-paroxysmal phenomenon, a form of acute mania succeeding to epileptic attacks of a severe character; under the present heading, reference is made to a state of frenzy, often of intense violence and acuteness, occurring in the inter-paroxysmal periods in some epileptics. It may last from a few minutes up to several hours

[1] Colman, *Lancet*, August 28th, 1903.

[2] See M'Carthy, *Journal of Nerv. and Mental Disease*, 1900, p. 144. Schultze, *Zeitsch. f. Psychiatrie*, 60, p. 795. Janet expresses the opinion that prolonged automatism is more likely to be hysterical than epileptic.

or days, in this respect differing from ordinary acute mania, which persists for weeks or months. The patient either passes suddenly into a condition of acute delirious excitement, or the onset of the attack may be preceded by prodromal symptoms, such as headache, dyspepsia, and hallucinations. The return to consciousness is nearly always sudden, sometimes through a convulsive seizure; and after it is over the patient has no recollection of what has happened, or at the best only a faint remembrance of the event. The following is an account of such an attack given by Dutil:[1] "The attack comes on suddenly, the patient becoming irritable and talkative. Then ensues frenzy of a vehement character. His violence and strength increase as resistance is made to control him. In the midst of this, he nevertheless appears to see and observe those who are about him, and gives to questions an appropriate reply. His actions are violent, but ofttimes very accurate. Under the influence of hallucinations, he strikes severely and wounds or injures himself. After a few minutes or some hours' duration, exhaustion ensues, and the attack subsides either suddenly or by degrees; when he wakes up there is no recollection of what has happened. If the attack has been of short duration, there may be slight rise of temperature; if prolonged, it may rise to 40° or 41° C. The attack is always followed by a period of lethargy and sleep, which in some cases may terminate in coma and death."

Attacks of this character may rightly be regarded as epileptic psychical equivalents, for phenomena of a similar kind may occur as post-convulsive psychoses. The intensity of the maniacal fury is one of the features of their epileptic nature.

All such attacks, however, are neither so prolonged nor so delirious in appearance as that just described. A minor form of epileptic mania may be seen in the *epileptic impulsions*, attacks characterised by the suddenness of their onset and their independence of external surroundings. To them the term "petit mal intellectuel" has been applied by Falret. Like the other forms of psychical equivalent, they are not confined to epileptics, but are of frequent occurrence in mental defectives and moral delinquents.

According to Féré, the prodromal symptoms are irritability, suspiciousness, and intolerance with fears and apprehensions;

[1] Dutil, *Traité de Pathol. Mentale*, Paris, 1903, p. 851.

others may have definite sensory and sensorial warnings (visual and auditory); or there may be head sensations of a distressing character. The impulsion occurs like a "discharge," and is followed by depression, sleep, and a state of exhaustion. During the attack the patient may answer questions, but there is no recollection of the acts accomplished during it. But there are variations from this type, in which the patient appears to be aware of the commitment of the act, and to preserve a faint remembrance of it. A striking feature of the impulsions lies in their similarity in the same person, and the regular periodicity of their recurrence.

The chief epileptic impulsions are: suicide, homicide, and pyromania; but there are many others, some of which are of a bizarre and indecent type, such are theft, gluttony (Féré), and exhibition. Some of the perverted sexual acts would seem also to belong to the same category.

In association with the clinical features just described, there is observed in these patients obvious evidence of structural stigmata of degeneration, as well as those mental stigmata to which reference has been made. The latter are seen chiefly in backward or defective mental development, in a tendency to hypochondriacal feelings and obsessions, and in a heightened susceptibility to the influence of even small quantities of alcohol (Bonhoeffer).

The epileptic impulsions are not infrequently carried out after an alcoholic outburst, during which the patient has partial retention of memory of what he has done, but seems to be impelled by some irresistible force to effect it.

When the attack is over, he frequently presents the after-stage of exhaustion, common to most epileptic seizures, during which he sleeps and from which he wakes like one who has had an ordinary epileptic fit.

Although in these cases a personal history of epileptic seizures is rare, there is often a highly neuropathic family history, or evidence of convulsions during infancy or early childhood.

3. **Dream States.** Under this heading are included psychical states of a paroxysmal and recurring character. The one to which reference is specially made is common to all psychopathic subjects, and is chiefly characterised by symptoms of a psychasthenic order. Such symptoms may be defined as epileptic psychical equivalents, when they conform to the features

already laid down as pathognomonic of epilepsy. The dream states are therefore described as psychical equivalents; for it has been shown that psychasthenic symptoms are occasionally seen as the prodromal psychoses of epileptic fits, although their occurrence as post-convulsive mental states is unusual.

Mention has already been made in an earlier chapter of the "dreamy states" described by Hughlings Jackson as a symptom in some cases of epilepsy, characterised by an aura of smell and taste, or with epigastric warning. The "dreamy state" sometimes consisted of a feeling of reminiscence; at other times the sensation was that of abject fear, and sometimes of impending dissolution. It is, however, important to distinguish between these conditions, which are really warnings of epileptic fits, from those which are described in this section. The latter refer rather to interparoxysmal phenomena and equivalent states than to "aura" symptoms. Sufferers from this condition describe the mental state as a feeling of non-existence, with doubts as to the reality of everything, even as to the material existence of their own bodies; they also have doubts as to whether what they see and hear are real. Such symptoms are frequently associated with others of an obsessional character, such as intense fear of sleeping alone, or fears and doubts as to whether they can meet and talk to certain persons, the whole picture being accompanied by intense anguish, and occasionally by terrible thoughts.

Although these symptoms may occur as warnings and prodromata of epileptic fits, they are more commonly met with as psychical states occurring in the interparoxysmal periods. Crichton-Browne[1] and A. Pick[2] have also described them as eventually becoming permanent phenomena in some cases of confirmed epilepsy. It has been observed that these symptoms occur more particularly in association with the catamenial periods, in this respect conforming to what is not uncommonly noticed with other psychical equivalents.

It has been stated by Pick, in his account of the dreamy mental states, that as these attacks become more permanent, the ordinary epileptic seizures assume a diminished frequency, a phenomenon which is also characteristic of other psychical

[1] Crichton-Browne, "Dreamy Mental States," *Lancet*, p. 1., 1895.

[2] A. Pick, *Brain*, 1903, p. 242.

equivalents. It is no uncommon thing to find an epileptic deploring the fact that his fits are being, or have been, replaced either by psychasthenic states, psychical attacks, or cephalic sensations, as the mental torture of the equivalent states far surpasses anything produced by the convulsive seizures.

The close relation existing between psychasthenic states and epilepsy has been described by P. Janet,[1] who, although he does not identify the two conditions, has stated that their phenomena can replace each other. He has introduced the term "Psycholepsy," to indicate the altered consciousness, which gives rise to the psychasthenic phenomena of the "dream-states" just described.

These symptoms, when they occur as psychical epileptic equivalents, may be preceded by prodromal symptoms, or they may, in turn, form the precursory phenomena, which terminate in a fit.

The following case is recorded to show the substitution of attacks of periodic headache for those of a psychical character, and the occasional replacement of the latter by psychasthenic equivalents, as just described.

The patient was a middle-aged single woman, who had suffered since puberty from severe periodic headaches, or "bilious" attacks, at or about the catamenial period. When she was *aet.* 39, these gave place to the phenomena about to be described. She presented the "facies epileptica," but no other stigmata of degeneration were observed. Her mother had suffered, after the birth of the patient, from a prolonged obsessional attack with suicidal feelings. At the age of 39, after some prodromal malaise and severe headache, and an aura of giddiness, accompanied by "twitching of the eyelids" on the right side, she lost consciousness, but did not fall, and does not seem to have had any convulsions. During the attack she walked some miles, having periods of partial return to consciousness, which she remembered afterwards. On recovering eight hours later she had complete loss of memory of her name and abode, and in consequence was taken charge of by the police. This attack was followed by severe headache, vomiting, and mental confusion, which lasted for several days. Since the original attack three years ago,

[1] P. Janet, "*Les Obsessions et la Psychasthenie*," Paris, vol. i.

she has had five other similar seizures, four of which were slight, not lasting more than three or four hours; but they were all proeeeded by prodromal malaise, "flickering" of the right eyelids, and were sueeeeded by vomiting, headache, and apprehensions of a "terrific character." These attacks immediately succeeded the catamenia, which were accompanied by great pain.

On several occasions, at the monthly period, in place of one of the attacks just deseribed, she has had spells of a profoundly psychasthenic character, with the phenomena of the dream state, in which she was unable to comprehend the reality of things and faces around her; she had terror of going into a room by herself, and was so apprehensive that she could not sleep alone. These attaeks were of a temporary eharaeter, and left her in her natural mental condition. She has taken large quantities of the bromide salts, from time to time, without benefit.

The case is instructive, in that it illustrates three kinds of paroxysmal attack, the most important of which is a psychomotor seizure, resembling in its features the attacks of epileptic wandering, already described. At the climacteric these attacks, on the one hand, replaced periodic headaches, from which the patient had suffered for many years; and on the other hand, were themselves replaced by occasional equivalent states of a psychasthenic character, the chief symptoms of which were those of the "dreamy mental state."

The epileptic features of the psychomotor seizures were an irregular periodicity, a constant motor warning, and the post-paroxysmal symptoms of vomiting, headache, and temporary mental confusion.

4. **Transitory delusional states** are frequently seen as inter-paroxysmal phenomena, and form a not uncommon variety of psychical epileptic equivalents. They are comparable to similar states, which follow epileptic convulsions, more particularly when these oeeur in series. Delusional symptoms are also repeatedly observed during the resolution from acute epileptic dementia, preliminary to the return of mental health after a serial outburst, or the status epilepticus. The attaeks are eharaeterised by delusional insanity of a mild type; there is rarely any fixed idea, the delusions rapidly disappearing after a few days. During this period the patient is irritable, resents interferenee, and is not

uncommonly pugnacious and difficult to manage; he is also confused, and refuses to carry on his usual avocations. There is frequently also lethargy and a loss of interest in his surroundings.

The attacks, which may last a few days, suddenly pass away, the patient having no recollection of what has happened. They may, or may not, be ushered in by prodromal symptoms. In one case these took the form of abnormal appetite, amounting to gluttony.

5. **Catatonic stupor.** I have records of three cases in which this condition was believed to represent a psychical epileptic equivalent. In one patient it was a post-paroxysmal phenomenon on one occasion, succeeding to short series of severe fits, but on another occasion there was no obvious antecedent convulsion. One of the attacks lasted four days, and the other one day. In another case the attacks of catalepsy only occurred as interparoxysmal symptoms.

Clinically this form of stupor, when observed in epileptics, does not differ from that occurring in non-epileptic persons. The facial expression is blank, fixed, and staring, while the general attitude is that of rigidity, and all attempts at passive movements of the limbs are met by resistance, the muscles being in that state which permits of the limb remaining in whatever position it may be placed. Similarly the jaws are clenched, so that it is difficult to get food into the mouth. "Mutism" also is a prominent symptom, articulation being for the time in abeyance, or limited to a few inarticulate sounds. The patient requires to be attended to, and has to be dressed and undressed. There is no recollection of the attacks, the onset and disappearance of which are sudden.

As states of a stuporous character, but without the striking phenomena of catatonia, have also been seen in the interparoxysmal periods without evidence of an antecedent convulsion, and as these conditions are not at all uncommon as post-convulsive symptoms, they have been grouped and described here as occasional psychical epileptic equivalents.

6. **Cephalic and "Aura" Sensations.** These include a number of interparoxysmal symptoms which are not true equivalents, but to which attention should be drawn, as many instances of abnormal transitory phenomena are recorded, whose

position in the hierarchy of epileptic manifestations is not superficially obvious.

Of the cephalic sensations, *headache* is without exception, the most common. Its relation to epilepsy and epileptic fits is discussed on a later page (p. 156), but reference is made to it here, as there is reason to believe that it may form an occasional epileptic equivalent. The most common association is to be found in the replacement of headaches in early life by epileptic fits in the later years; but the converse is also seen. Moreover, one finds sometimes in cases of so-called arrested epilepsy, the continuance of attacks of paroxysmal headache. This latter condition should, however, be regarded rather as the substitution of periodic headaches for epileptic seizures.

Under the designation of "*aura sensations*" are included a number of psychical and sensory phenomena, occurring in the interparoxysmal periods, which have a colourable resemblance to epileptic equivalents, but which in reality are the auras, or warnings, of a fit, and form one of the varieties of the incomplete, or minor, seizure, already fully described. It has been shown on an earlier page, that the law of Herpin formulates the identity between the incomplete attacks and the initial phenomena of the complete seizure. In this way a ready explanation is at hand of some of those interparoxysmal symptoms, which have been regarded as epileptic equivalents. This is more especially referable to those fits which have a psychical warning; hence, such sensations as, "intense fear of something dreadful about to happen," or of the "dreamy state," occurring as an occasional and isolated interparoxysmal symptom, are not necessarily equivalents, but merely the initial phenomena of the complete attack.

A similar explanation holds good for attacks of pain, whether in the abdomen, giving rise to a "false appendicitis," or in the region of the heart, to a "pseudo-angina pectoris." In patients presenting these symptoms as isolated sensations, other attacks are usually found in which there is a "march" of the sensation, as well as complete fits. Among such conditions may also be placed the gastralgias, nausea, vomiting, and voracious appetite, all of which are known as aura and prodromal symptoms of complete epileptic seizures.

7. **Miscellaneous Equivalents. Substitution Phenomena.** A number of miscellaneous symptoms have been, from time to time, recorded as epileptic equivalents, more especially by C. Féré. Among these asthmatic affections, spasmodic laughter, and automatic whistling may be mentioned.

Brief reference will be made to a few abnormal conditions, which have come under the author's personal observation; but whether they should be regarded as epileptic equivalents, or substitution phenomena allied to epilepsy and occurring in the same patient, is a debatable point.

Narcolepsy, or periodic falling asleep, was a psychical condition occasionally observed. In one case, under my care, attacks of falling off to sleep, lasting from one to five minutes, were interchangeable with what appeared to be attacks of minor epilepsy; and in another, attacks of a narcoleptic character occurring by day, were associated with fits during sleep. Féré[1] considers that there is a narcoleptic epilepsy, distinguished by attacks of deep sleep, occurring as equivalents of fits. They are usually found between the epileptic seizures.

The existence of a genuine narcolepsy of epileptic character, occurring as a psychical epileptic equivalent, seems indisputable, but it would also appear to be a symptom of hysteria.[2] It should not be forgotten that a tendency to fall asleep is a symptom of cerebral syphilis.

I have observed one case of *paroxysmal laughter*, frequent attacks of which occurred as interparoxysmal phenomena.[3] The attacks were of short duration, only lasting a few seconds; they came on without any extraneous cause of merriment, and upon their subsidence the patient was unaware of what had happened. They appeared to be of the nature of transient attacks of psychical epilepsy. The patient had a left-sided infantile hemiplegia, and an occasional prolonged attack of unilateral convulsions.

There was one case, in which attacks of *paroxysmal sneezing*, occurring two or three times a week for a number of years, both winter and summer, preceded the onset, at forty years of age, of genuine nocturnal epileptic fits. The patient had 8 dioptres of hypermetropia in one eye, the other being

[1] Féré, *Rev. de Médecine*, 1898, p. 430.

[2] M'Carthy, *Amer. Journ. Med. Sciences*, vol. 119, p. 178.

[3] Oppenheim, *Rev. of Neurology*, 1903, p. 277.

emmetropic; no other stigmata were observed. A paternal uncle had a fit, in which he died. These attacks are more probably substitution phenomena than epileptic equivalents, having the same relation to epileptic seizures as attacks of periodic headache, in that they disappear when the epileptic fits commence.

IV. THE PERMANENT INTERPAROXYSMAL MENTAL CONDITION. EPILEPTIC DEMENTIA.

The mental symptoms, described under this heading, are more or less constant during the intervals between the attacks, and constitute the true epileptic mental condition. These changes include all degrees of mental impairment, from slight interference with memory up to the most pronounced forms of chronic dementia.

A brief reference may first be made to the proportion of epileptics, who become mentally afflicted, according to the statements of different writers. Esquirol,[1] for instance, stated that of 385 epileptic women observed in the Salpetrière, 73 per cent. were mentally afflicted. Russell Reynolds[2] referred to the fact that of 62 cases studied by himself, 38 per cent. were free from any mental failure, 46 per cent. showed some degree of mental impairment, and 14 per cent. were demented. Wildermuth[3] mentioned that the percentage varied according to whether the cases were observed inside or outside an institution. From the observation of his cases, he found 78 per cent. mentally afflicted inside, and 47 per cent. outside the institution.

The segregation of epileptics in special institutions, which has been customary during the past two or three decades, has permitted of a fuller study of the interparoxysmal mental conditions; and from the observations of several recent writers the following facts have been collated:

Habermaas,[4] who has studied the mental condition in detail amongst the patients attending the Epileptic Institute at Stettin, gives the following figures: 17·3 per cent. were intellectually normal, 21 per cent. were able to earn wages, 30 per cent. could do some work, and 49 per cent. were so demented, that they

[1] Esquirol, *Des Maladies Mentales*, etc., Paris, 1838.

[2] Russell Reynolds, *Epilepsy*, London, 1861, p. 43.

[3] Wildermuth, quoted by Habermaas.

[4] Habermaas, *Allg. Zeitsch. f. Psychiatrie*, vol. lviii. p. 243.

were unable to work at all. Of those patients who were inmates of the Institution, only 13 per cent. were intellectually normal, and 22 per cent. were demented, while the remainder showed intermediate degrees of mental failure.

In the Wuhlgarten (Berlin) Institute for Epileptics, Bratz[1] was able to classify the epileptics, according to their mental condition, into the following subdivisions: (*a*) the mentally normal, 15·8 per cent.; (*b*) the mentally disabled, who were fit for work, 33 per cent.; (*c*) the mentally disabled, who were unfit for work, 50·9 per cent.

In the Craig Colony, New York State, the following classes of epileptics and their percentage frequency have been described: (*a*) mentally good, 17 per cent.; (*b*) mentally "fair to feeble," 67 per cent.; (*c*) demented (including imbeciles and idiots), 15 per cent.[2]

Following upon the lines laid down by Reynolds, Habermaas, and others, I have subdivided epileptics, according to their inter-paroxysmal mental condition, roughly into four classes, showing slight, profound, or intermediate degrees of mental deficiency, and the general features of those classes may be briefly described as follows:

Class A. In this division are included all those epileptic individuals in whom no mental impairment can be detected. They may be regarded as differing in no way from normal persons in the same social sphere and with similar educational advantages. The memory is good; they are bright, active, and intelligent, and are capable of earning their own living; only in rare instances do they show the epileptic face. Their physical condition is good, and for all practical purposes may be regarded as normal.

Class B. This class, to which the majority of epileptics belong, includes those who exhibit the first degree of mental enfeeblement, which is characterised mainly by some defect of memory, more especially a forgetfulness of recent events. In other respects their mental condition is good; they have fair intelligence and capacity for work, they are able to earn their living and to attend to their several duties. Their physical condition varies. A considerable proportion show the epileptic face.

In *Class C.* are found those cases which present the second degree of mental impairment. In addition to an impaired memory, there is defective power of initiation and capacity

[1] Bratz, *Neurol. Centralbl.*, 1905, p. 611. [2] Spratling, *Epilepsy*, 1904, p. 299.

for work, which, however, may be well done under direction and supervision. They are slow in comprehension, and are often lazy. Many of them are irritable, eccentric, and passionate. About 50 per cent. show the epileptic face.

Class D. contains those who show the third or most pronounced degree of dementia. They present the typical features of epileptic dementia, viz., a defective memory, confusion of ideas, poor capacity for work even under direction, absence of initiative, and a slow and dull comprehension. Although not legally insane, they require supervision. Their physical condition is as a rule good, they eat heartily and sleep well. The majority have the epileptic face.

The several varieties and degrees of the interparoxysmal mental state in epilepsy, and the relation to the various stages and types of the disease, will be discussed in detail under the following headings:

1. Sex.
2. Hereditary predisposition to epilepsy and insanity.
3. Duration of the disease from the commencement of the convulsions.
4. The age of the patient at the onset of the disease.
5. The character, type, and combination of the seizures.
6. The frequency of the seizures.
7. The facies epileptica.
8. Stigmata of degeneration.

1. *Sex.* In this and the following tables, epilepties have been classified according to the degree of mental impairment, already described, into four subdivisions.

Table 22, *giving the total number of males and females with their percentage frequency in the four sub-divisions of mental failure, from* 161 *cases of Epilepsy at the Chalfont Colony.*

Mental Class.	Males.	Females.	Total.	Percentage.
A	9 (8·8 %)	13 (22·0 %)	22	13·6
B	35 (34·3 „)	16 (27·2 „)	51	31·6
C	29 (28·4 „)	12 (20·3 „)	41	25·4
D	29 (28·4 „)	18 (30·5 „)	47	29·1
Totals -	102	59	161	99·7

From this it appears that the greater number of epilepties (31·6 per cent.) were found to belong to Class B, and to show

only the slightest or first degree of mental impairment. But if, on the other hand, the table be so subdivided that Classes A B and C D be bracketed together, it will be found that 45 per cent. belong to the former, who show none or only slight mental deficiency, while 54·5 per cent. belong to the latter and include those with mental deterioration in its most pronounced forms. It is therefore obvious that the cases herein tabulated give a larger proportion which exhibit mental deterioration of a marked type.

It should, however, be pointed out that the above percentages do not faithfully depict the mental state of epileptics in general, as it is obvious, that those cases which seek treatment at a colony for epileptics do so on account of some mental disability, which prevents them earning a living under ordinary conditions. Secondly, if the table be read in vertical instead of horizontal columns, it will be seen that a larger percentage of women (22 per cent.) escape the deteriorating influence of epilepsy than men (8·8 per cent.); but that when dementia supervenes and reaches its most pronounced form, a larger percentage of women (30·5 per cent.) are affected than men (28·4 per cent.). The most frequent mental condition in males is seen to be a slight impairment of the memory and a blunting of the higher mental faculties (Class B).

If these figures are compared with those given by Habermaas Bratz, and others, on a previous page, a close correspondence is found to exist between them, as will be apparent from the annexed table:

Table 23, *showing the degrees of mental impairment and their percentage frequency from several Institutions for Epileptics.*

Mental State.	Chalfont.	Stettin.	Wuhlgarten.	Craig Colony.
	Per cent.	Per cent.	Per cent.	Per cent.
Mentally normal -	13·6	13	15·8	17
With poor memory -	31·6	65	33	67
Feeble minded - -	25·4			
Demented - - -	29·1	22	50·9	15

2. *Hereditary disposition.* The hereditary tendencies referred to under this heading are epilepsy and insanity. Of the total of 161 cases there was no note of a heredity to either epilepsy or insanity in seventy-three. The following table shows the

result of the investigation among the remaining eighty-eight cases:

Table 24, *showing the presence or absence of a hereditary disposition in eighty-eight cases of confirmed Epilepsy.*

	A.	B.	C.	D.	Total.
No known heredity - -	11	18	9	11	49
Epilepsy { Paternal - -	0	8	2	3	29
Epilepsy { Maternal - -	4	5	5	2	
Insanity - - - -	1	2	2	5	10
	16	33	18	21	88

The facts, which may be deduced from Table 24, are that those epileptics who show no mental impairment, or only some impairment of memory, present a lower percentage with hereditary neuropathic tendencies than those who exhibit dementia in its more pronounced forms. This fact, taken by itself, points to the deteriorating influence, which a family disposition to epilepsy or insanity has upon the mental condition of those who subsequently develop epileptic seizures.

The table may profitably be compared with one, which is given upon a later page, to illustrate the effects of a hereditary history upon the general results of treatment in epilepsy. From this it is seen that of those cases which have a hereditary disposition to epilepsy a larger percentage became confirmed epileptics than those without such history. (Table 31, p. 214.)

The general deduction, therefore, which may be drawn from the above facts is that *a family tendency to either epilepsy or insanity, although offering no obstacle to the arrest of the seizures in favourable cases, materially increases the probability of the disease becoming confirmed and the supervention of dementia.*

3. *Duration of the disease.* The influence of the duration of the disease upon the degree of mental decrepitude has been regarded by many writers as of much significance. In opposition to the commonly expressed opinion upon this matter, Russell Reynolds held to the belief that the duration of epilepsy was without influence upon the mental condition of the epileptic.

A table has, therefore, been constructed with a view to throw further light upon this part of the subject.

Table 25, *showing the duration of the disease and the number and percentage frequency of the cases in the four mental classes.*

Duration.	A.	B.	C.	D.	Total.
Under 5 years	6 (27·2 %)	10 (45·0 %)	3 (13·6 %)	3 (13·6 %)	22
6 to 10 „	13 (30·9 „)	10 (23·8 „)	7 (16·6 „)	12 (28·5 „)	42
11 „ 15 „	1 (3·1 „)	10 (31·2 „)	10 (31·2 „)	11 (34·3 „)	32
16 „ 20 „	1 (3·5 „)	4 (14·2 „)	10 (35·7 „)	13 (46·4 „)	28
21 „ 30 „	1 (4·7 „)	8 (38·0 „)	4 (19·0 „)	8 (38·0 „)	21
Over 30 „	1 (6·6 „)	6 (40·0 „)	7 (46·6 „)	1 (6·6 „)	15
Mean duration	3·8 years	8 years	6·8 years	8 years	160

From a consideration of the above table the following general deductions may be made:

(1) That under five years' duration there is a considerably greater percentage of cases with no mental impairment, or merely some interference with the memory than of those with well-marked mental deficiency.

(2) That of those cases in which the disease has existed from six to ten years there is a slightly greater percentage in Classes A and B than in Classes C and D.

(3) But that if the convulsions have lasted over ten or eleven years only a trifling percentage are found in Class A, the majority showing the mental characteristics of Classes C and D.

(4) It should, however, be especially pointed out that in a very few cases the disease may have lasted for periods of thirty or more years without any obvious mental impairment.

It may, therefore, be concluded that *although the duration of epilepsy from the commencement of the seizures is a potent factor in determining the subsequent mental condition, it is not the only influence in the production of dementia.*

4. *Age at onset.* There was a total of 140 cases in which the age at onset of the disease was known.

Table 26, *showing the age in quinquennial periods up to twenty years at onset of fits, with the number and percentage frequency in the four mental classes.*

Age.	A.	B.	C.	D.	Total.
Birth to 5 years	1 (3·3 %)	9 (30·0 %)	9 (30·0 %)	11 (36·6 %)	30
6 „ 10 „	3 (11·5 „)	8 (30·7 „)	7 (26·9 „)	8 (30·7 „)	26
11 „ 15 „	10 (18·1 „)	15 (27·2 „)	17 (30·9 „)	13 (24·5 „)	55
16 „ 20 „	7 (24·1 „)	8 (27·6 „)	5 (17·2 „)	9 (31·0 ,)	29
					140

From this table we may draw the following general conclusions:

(1) Those epileptics in whom the disease commences in early childhood, show only a small percentage with normal mental health and a high percentage with profound mental impairment.

(2) From the first quinquennial period onwards the percentage of cases showing no obvious mental deficiency progressively increases, until in the fourth quinquennium (16 to 20 years) a greater percentage is found with normal mental health than with marked mental disability.

No general deductions may be safely drawn from the cases in the succeeding quinquennial periods owing to the fewness of the cases observed, but it may be said in general terms that, in epilepsy commencing over twenty-one years, about an equal percentage show impairment of memory and the severer degrees of mental deficiency.

These results are at variance with what was noticed by Russell Reynolds working along similar lines. This observer noted that, whether the disease commenced early or late in life, more cases were to be found in Classes A and B than in C and D; in other words, "that the mind is not specially affected in those whose epilepsy begins early, neither is it in those whose disease is late in its development.[1]

On the other hand, the results obtained in this present research support the contention of most of the older writers on epilepsy, and confirm what is pointed out in the chapter upon general prognosis, viz. *that epilepsy commencing in infancy and childhood is least favourable for arrest of the fits and most favourable for the production of confirmed cases.* It is also found that the common type of epilepsy—that commencing during the period of puberty—is most suitable for arrest of the seizures and least likely to be associated with mental infirmity.

5. *Character of the fits.* The character of the epileptic seizures has long been regarded as an important factor in determining the mental condition. It is generally accepted that the minor type exerts a greater deteriorating influence upon the mind than the convulsive or major type. This is no doubt the common experience of those through whose hands numbers of epileptics

[1] R. Reynolds, *Epilepsy*, p. 166.

pass. A table has therefore been constructed to show the effect of the two common types of seizure upon the mental state. A total of 159 cases has been used, in whom the character of the fits was definitely known.

Table 27, showing the percentage frequency of the four mental classes in the common types of Epilepsy.

Type.	A.	B.	C.	D.	Total.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Major type only	24·3	27·0	27·0	21·6	74
Minor type only	7·1	42·8	21·4	28·5	14
Combined major and minor types	4·2	32·8	25·3	38·9	71
					159

The major type, therefore, when occurring alone, is not found to be characteristically accompanied by profound mental impairment, for the table shows an almost equal percentage of cases in the several mental classes. On the other hand, the greatest frequency and the highest degree of dementia is found in those cases which are characterised by a combination of the major and minor attacks.

The general deductions therefore are:

(*a*) Mental deterioration is found in association with both types of seizure, but is less frequent in those cases in which the major fit is the main expression of the disease; (*b*) freedom from mental impairment is also found in both types, but to a small extent only in those cases characterised by the minor fit, whether alone or in conjunction with the major fit; (*c*) the mind is more frequently affected to a slight extent in those cases in which the minor seizures occur alone; (*d*) the mind attains its most universal and profound impairment when the disease is manifested by a combination of the major and minor attacks.

6. *Frequency of the seizures.* In an institution for epileptics it is a comparatively easy matter to register the number of seizures, and this has been done on especially constructed charts (see Charts), so that at a glance there can be seen daily, weekly, monthly, etc., incidence of the seizures, whether major or minor in character, and whether they occur by day or during sleep.

Table 28, *showing the percentage frequency of the attacks in the four mental classes.*

Frequency.	A.	B.	C.	D.	Total.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Daily (one or more)	0·0	17·4	27·9	55·5	18
Weekly (one or two)	1·3	26·3	33·3	39·0	72
Monthly (one or two)	25·7	48·5	17·1	8·5	35
Quarterly (one or two)	57·1	14·2	28·5	0·0	7
Yearly (one or two) -	50·0	50·0	0·0	0·0	8
In series - - -	7·6	23·0	23·0	46·1	13
Arrested cases - -	50·0	16·6	16·6	16·6	6
					159

At the first glance there will be noticed a definite relation between the frequency of the seizures and the mental state, to such an extent that when the fits are of very frequent recurrence (daily) none of the patients were without obvious mental impairment, over 50 per cent. of them exhibiting the deepest degree of dementia. On the other hand, if the seizures are so infrequent as to be counted by the quarter or the year, over 50 per cent. were found to be mentally normal, or merely with defect of memory. Between these extremes there are various gradations, so that the general statement may be made that *there is a direct relationship between the frequency of the seizures and the degree of mental impairment*—the more frequent the attacks the more common and profound the associated dementia.

If attention is now directed to those seizures, whose recurrence is characterised by series, it will be observed that in this type we are dealing with cases unfavourable to the maintenance of mental integrity, considerably more than double the number of such patients being found with the severer grades of mental deficiency. In this type of epilepsy, therefore, we are confronted with a variety of the disease prone to the development of dementia.

The mental state of those cases in which the seizures have been arrested, either with or without treatment, presents features of great interest, for the table shows, that even when the fits are arrested, there is not necessarily an unimpaired mental state.

7. *The facies epileptica.* The close relation which exists between mental disability and facial expression is nowhere more readily seen, and studied, than in cases of confirmed epilepsy. So characteristic indeed is the facial appearance of many epileptics that the term 'facies epileptica' has been applied to it.

Although difficult to define, it is readily discerned by those who are in the habit of treating large numbers of epileptics. It may, in general terms, be described as an expression of dulness and heaviness, with an absence of emotional mobility of the features. It differs from that of the ordinary dement by a particular expression, which stamps the individual as an epileptic.

That the epileptic appearance is largely dependent upon the mental attitude may be seen from the following table, which has been constructed to show the percentage frequency of the 'facies epileptica' in the four mental classes.

Table 29, *showing the frequency of the facies epileptica in the mental classes.*

	A.	B.	C.	D.	Total.
Total Cases -	22	51	41	47	161
Facies epileptica seen in -	2 or 9·0 per cent.	16 or 31·3 per cent.	21 or 51·2 per cent.	34 or 72·3 per cent.	73 or 45·3 per cent.

Of a total, therefore, of 161 cases, seventy-three, or 45·3 per cent., showed the expression of countenance described as the 'facies epileptica.' Its presence was noted in those with mental integrity as well as in those showing the several degrees of dementia; but its percentage frequency showed a progressive accession as the degrees of dementia increased, so much so that amongst the most demented, 72·3 per cent. showed the characteristic expression. It is more frequently seen in male than in female epileptics, for of the seventy-three cases only nineteen were women. (See also p. 162.)

8. *Stigmata of degeneration.* It would be a point of interest to ascertain whether the degree of mental impairment stood in any relation to the frequency of degenerative structural stigmata.

With this object in view a table has been constructed to show the presence, or absence, of stigmata in 100 cases of confirmed epilepsy, and their relation to the four classes of mental impairment.

Table 30, *showing the numerical frequency of neuropathic stigmata in the four mental classes in one hundred epileptics.*

Mental State.	With Stigmata.	Without Stigmata.
A	2	7
B	19	12
C	26	5
D	27	2

If the groups which contain the slightest degrees of mental impairment are classified together, as well as those containing the most marked forms of mental enfeeblement, it will be evident that the former contain an almost equal percentage of cases with and without stigmata, while the latter contains 53 per cent. with, and only 7 per cent. without, stigmata.

From this it is apparent that there exists a close relation between the presence of neuropathic stigmata and the degree of mental impairment: *for the more pronounced the mental enfeeblement the more frequent the evidence of structural degeneration.*

This is one of the most important arguments in favour of the view that the interparoxysmal mental state in epilepsy is an integral feature of the disease, and, as such, is a prominent symptom of the malady.

Is the Permanent Interparoxysmal Mental Condition in Epilepsy an Integral Feature of the Disease?

It is a well recognised fact that both mental integrity and mental impairment may be found amongst epilepties; but the question arises whether the mental condition, which characterises the later interparoxysmal periods, is an integral feature of the disease, or is induced by the character, the duration, and the frequency of the seizures, or by any other peculiarity of the malady.

In the first place, it may be stated *that mental failure is not essential to the idea of epilepsy* (Reynolds). On the one hand, it is seen that a certain number of epilepties present no obvious mental deficiency, notwithstanding an inherited predisposition to the disease, a duration of the malady from twenty to thirty years, its onset in early life, the combination of both the major and the minor seizures, and a recurrence of the seizures so frequent that they may be counted by the week. On the other hand, it has been shown that pronounced dementia may exist when there is no known heredity, when the disease is of short duration (under five years), when the age at onset is as late as the fourth decennium, whether the attacks are of the major or minor type, and even when the seizures have been completely arrested.

Secondly, the deduction may be made that *the mental condition is not solely a consequence of the seizures, but is an expression of*

the same nervous constitution which gives rise to the convulsions; for the following reasons:

(*a*) A hereditary disposition to epilepsy or insanity favours the production of the severer types of dementia and the establishment of the disease as a confirmed malady.

(*b*) The duration of the disease, though a potent factor in determining the mental condition, is not the only cause, for some cases of dementia are met with when the disease has lasted less than five years, and others with mental integrity after the fits have persisted for over fifty years.

(*c*) Upon the character or type of the seizures depends to some extent the degree of mental impairment. On the one hand, the major convulsions are as commonly accompanied by mental health as by profound dementia; on the other hand, the minor seizures are more frequently found with mental failure than with mental health. But the character of the seizures, though influential, is not solely responsible for the mental condition, for it is found that both types, separately or in combination, are associated with mental integrity as well as with mental deficiency.

(*d*) The *facies epileptica* is found in those who show the slighter as well as the more profound degrees of dementia; but there is also a small percentage of epileptics with normal mental attitude in whom this feature is present. Although its existence is not directly attributable either to the degree of mental infirmity, or even to any mental deficiency at all, the proportion of cases in which it is observed, is in direct relation to the intensity and severity of the mental decrepitude.

(*e*) There is a direct association between the frequency of the seizures and the mental state, for the more frequent the attacks the greater the degree of mental impairment, and *vice versâ*. If the severity of the disease in any given case is gauged partly by the frequency, and partly by the character-combination of the paroxysms, it is found that the more severe the disease the greater the tendency towards dementia.

(*f*) The age at which the disease commences influences to some extent the subsequent mental condition. The evidence brought forward tends to support the view that *the earlier the onset of the epilepsy, especially under ten years of age, the greater the probability of mental failure, and the establishment of the confirmed malady.*

(*g*) The presence of structural stigmata of degeneration, indicating a strong hereditary degenerative disposition, presents an important argument in favour of this view, for it has been shown that in those epileptics, in whom there existed marked mental enfeeblement, there was a high percentage who exhibited structural stigmata. Those cases also in which the disease commenced between birth and five or ten years of age presented a larger percentage of stigmata than at subsequent ages, a fact which confirms the already expressed opinion that *the epilepsy of infancy and childhood is the most favourable for the development of the confirmed disease and most likely to show the higher grades of mental enfeeblement.*

CHAPTER VII.

MISCELLANEOUS PHENOMENA OF EPILEPSY.

1. Neuropathic associations — Paroxysmal headache — Chorea — The tics — Myoclonus epilepsy—Chronic nervous diseases.
2. Accidents due to epileptic fits—tongue biting—scalp wounds—fractures and dislocations—ecchymoses—burns and scalds.
3. Physical condition of epileptics—facies epileptica—general functions—body weight—tremor—cardiac and pulmonary diseases.

NEUROPATHIC AND OTHER NERVOUS ASSOCIATIONS OF EPILEPSY.

Epilepsy, being an inherited degenerative malady, is not infrequently found in association with other functional disorders, which have a similar predisposition and degenerative foundation. The co-existence of epilepsy with these afflictions is seen both in the patient and in the family history; one form of neurosis may be found in the parent, another observed in the offspring; or different types may exist in the same person at different periods of life, or may even be observed to occur simultaneously.

Epilepsy may co-exist with various forms of chronic nervous disease, such as tabes dorsalis, exophthalmic goitre, and paralysis agitans; and its association with the acute psychoses, such as mania, delusional states, the obsessions, and the perversions, is, as already explained, either in the nature of post-paroxysmal phenomena, or as psychical equivalents. It is therefore a matter of clinical interest to study the neuropathic associations of epilepsy, and to ascertain their character, their frequency, and the manner of their co-existence. These are:

1. Epilepsy and periodic headache.
2. Epilepsy and chorea.
3. Epilepsy and "the tics."

1. **Epilepsy and periodic headache.** Epilepsy and paroxysmal headaches are not uncommonly associated. In my experience the latter condition is less often the classical "hemicrania," or migraine, than the so-called "bilious" attack.

This association was observed in 4·3 per cent. of my cases of epilepsy, and was of the following nature:

(*a*) Periodic headaches in one parent, epilepsy in one of the offspring, or *vice versa*.

(*b*) Headaches in early life replaced by epileptic fits in later life.

(*c*) The replacement of fits in early life by headaches in the later years.

(*d*) The simultaneous existence of periodic headaches and epilepsy in the same person.

The most common association is without doubt the replacement of headaches in early life by epilepsy in the later years (*b*). When epileptic fits ensue, the headaches either cease altogether, or continue less frequently and in modified severity. In the cases in which this was observed the epileptic attacks commenced usually after middle life, and the deduction is not unnatural, that the epileptic seizures of the later years are really substitutes of the earlier periodic headaches; and in many cases of so-called senile, or late epilepsy, the previous existence of periodic headache is a feature of common clinical occurrence.

These conditions may be illustrated by the narration of two cases, in the first of which epilepsy was replaced by periodic headaches; and in the other, migrainous attacks in youth gave place to epileptic fits after marriage.

A male, clerk, *æt.* 36, complained of attacks of paroxysmal headache, lasting about three hours, and coming on usually in the early morning. He has had attacks of a similar character for ten or twelve years, occurring in series every week or ten days. He had epileptic fits from the ages of 16 to 25 years, but there has been no recurrence of the fits since the onset of the headaches, except on one occasion, when he was under strong emotion on hearing of the death of his mother. He has had two attacks of Herpes Zoster, one over the ophthalmic division of the right fifth nerve, the marks of which are seen, and another two years ago, over the right side of the chest. His father, now aged 66, is a confirmed epileptic, and a sister suffers from

paroxysmal headache. The patient presented no physical signs of nervous disease, and no obvious stigmata of degeneration.

A female, *æt.* 32, suffered from migrainous headaches (giddiness, loss of sight, headache and retching) from 12 to 29 years of age, when she married. Since marriage, three years ago, the migraine attacks have become less severe and less frequent, their place being taken by periodic headaches of a slight character. But, within the past fifteen months, she has had two epileptic fits, and recently some "giddy" sensations, but no repetition of headaches. The palate is high, but not deformed, and there are no physical signs of nervous disease. Her mother suffers from "faints"; a sister has "bilious" headaches, and has on several occasions fallen down in the attacks; another sister suffers from occasional headaches.

The close relation which exists between paroxysmal headache and epileptic fits, has led many writers to the belief that headaches may occasionally be the "equivalents" of epileptic seizures. It is not unlikely that this may be the nature of the relation in some instances; but I would submit, in view of the two cases which have just been recorded, that the migrainous, or "bilious," headache and epileptic convulsions are symptoms of two separate disorders, which are so closely related that they may replace each other. We have seen that an epileptic parent may have two children, one of whom suffers from headaches, and the other from epilepsy at one period of life, and headaches at another.

That epilepsy and migraine are, notwithstanding their close genetic relationship, two separate maladies, seems obvious from the fact that the mental states of the former, whether paroxysmal or interparoxysmal, are not observed in migraine. They may be combined as neurosal affections in the same family, and even in the same individual, and one may replace the other. Migraine is a curable malady, while epilepsy is so to only a small extent. Epilepsy and paroxysmal headache therefore are not identical, but they are interchangeable, the one being substituted for, or replacing the other, in the same person at different ages.

2. **Epilepsy and Chorea.** Epilepsy and chorea are frequently associated, but it would appear from my cases to be more usual for epilepsy to be mentioned in the family history of choreic patients, than the converse.

Chorea and epilepsy, and chorea and infantile convulsions, are also found, but less commonly, in the same patient.

It may be stated in a general way that the relations between chorea and epilepsy are of the following nature:

(*a*) Chorea is found in the family histories of some epileptics.

(*b*) Chorea is also found in the personal history of some epileptics—2 per cent.

(*c*) Epilepsy is not infrequently found in the family history of choreic patients—14·2 per cent.

It is obvious from a study of the literature of this subject, that an intimate relation exists between epilepsy and chorea. The facts just narrated show that chorea may predispose towards epilepsy, and epilepsy towards chorea, the latter being the more common in my experience. In a neuropathic family, epilepsy and chorea may be present in different members; for example, one child may be an epileptic and another have chorea: an alcoholic father had two children, one an epileptic, the other choreic. Epilepsy and chorea may be present either simultaneously, or at different times in the same person; in one such patient an attack of chorea was followed by freedom from fits for a year.

In this connection, reference is only made to chorea minor: but it is well known that choreiform jerkings, and 'tic-like' spasms may be observed in epilepsy, as well as in other degenerative neuroses. These are, however, not true chorea, and may be included more appropriately in the next group.

3. **Epilepsy and 'the tics.'—Myoclonus Epilepsy.** Considering that Epilepsy and 'the tics' are both expressions of an inherited degenerative tendency, their co-existence in the same person is notably rare. In the present series only two cases of this association were observed. They were both young women in adolescent life; both presented tic-like contractions of the muscles of the neck, body and limbs, and both had epileptic fits.

In one, *æt.* 26, there was a strong neuropathic history, a younger brother being blind, paralysed and epileptic, and the maternal grandmother was also blind and paralysed. The patient was mentally defective. The tic-like movements began when she was about two years old, and the epileptic fits when she was aged twenty-four.

The other patient, *æt.* 25, was a seven months child. She had her first fit when fourteen years of age, but the jerkings had been present from early childhood. The 'tic-like' movements involved the head and neck, both arms, but more especially the right, and the articulation was of a jerky character. She had a high and narrow palate, but there were no fits known in the family, although a cousin had "chorea," and a sister died of phthisis.

A variety of epilepsy, in which convulsive seizures are associated with myoclonic, or tic-like, jerkings of the muscles, has been described as *myoclonus epilepsy.* It has been defined as "an association disease, characterised by paroxysmal, asynchronous, bilateral, lightning-like contractions of the trunk and of the proximal muscles of the extremities with varying intervals of entire freedom from such movements, and accompanied by a more or less persistent grand mal type of epilepsy." (Spratling.)

It has been well described by Unverricht, Lundborg, and most recently by Clark and Prout,[1] and it is not unlikely that under this description may also be included such clinical states as choreic epilepsy, and epilepsy with multiple tics.[2]

It is an eminently hereditary malady, instances of it having been observed in several members of the same family. The onset of the malady occurs during early adolescence, and as a rule the epileptic attacks precede the myoclonic condition, the former being major fits, the latter sudden involuntary contractions of the muscles of the trunk and limbs.

4. **Epilepsy and Chronic Nervous Disorders.** Epilepsy may co-exist with various forms of nervous disease, associations which are of a purely accidental kind, and do not appear to have any influence upon the character, or course, of the malady. Thus, an epileptic from childhood developed locomotor ataxy in later life; another showed symptoms of paralysis agitans; in a third, symptoms of gumma cerebri were engrafted on the original epileptic fits; and two cases developed Graves' Disease, after epilepsy had been established in one case for five years, and in the other from infancy.

[1] Clark and Prout, *American Journal of Insanity*, 1902, No. 2, where a bibliography is given.

[2] See E. S. Reynolds, *Rev. of Neurology*, January 1906, a case of paramyoclonus epilepticus.

ACCIDENTAL RESULTS OF EPILEPTIC FITS.

The epileptic is prone to many accidental circumstances resulting from the sudden and severe nature of the attacks, which form the most alarming symptoms of his disease. Some of these accidents result from the intensity of the muscular spasm and contraction during the convulsion, while others are due to falls, knocks, burns, etc., received during the stage of unconsciousness. Apart from the serious character of many of these accidents and the fatal results which may ensue, their presence is often of value in diagnosis.

(*a*) Probably the most frequent and characteristic accident is *tongue-biting*, with its resulting scars, of either a temporary or permanent nature. The proportion of epileptics who 'bite the tongue' during the seizure has been stated by Spratling to be about 22 per cent.; but it should be remembered that tongue-biting depends entirely upon the character of the seizure, and its absence should not militate against the diagnosis of epilepsy, if loss of consciousness can be established on other grounds. The tongue is usually bitten during the clonic stage of the seizure, being caught by the forcible movements of the lower jaw. It is most commonly bitten about the junction of the anterior and middle thirds towards the tip upon one or both sides. Sometimes a piece is bitten out, at other times there is merely a superficial erosion. The resulting pain and discomfort may be severe, the wound thus produced being sometimes the seat of a troublesome glossitis. The insertion of an indiarubber ring or other object to keep the jaws apart is the best preventive.

(*b*) *Scalp wounds and injuries to the face and head.* These are amongst the common accidents resulting from the epileptic fit. As already mentioned, the fall of an epileptic is peculiarly constant, either backwards, forwards, or sideways. These lead to incised wounds of the scalp, and their repetition to marked cicatricial scarring and deformity. Falls upon the occiput are probably the most frequent, to such an extent indeed in some cases that the patient is advised to wear an occipital pad, so as to break the force of the fall. Falls upon the forehead are less common, but they produce deformity, more particularly through fracture, or dislocation of the nasal bones. Wounds and bruises may be found, however, on any part of the head, owing to accidental striking against pieces of furniture or other objects during the fall.

(*c*) *Fractures and dislocations.* Dislocation of the shoulder joint is one of the commonest accidents of this character. In one case, during the earlier stage of the disease the right shoulder, and later on both shoulders, were dislocated during each fit. Once such dislocation has occurred its repetition is facilitated. Various methods have been devised to prevent its occurrence, such as the mechanical support of a specially adjusted jacket; but in cases in which this accident happens repeatedly, the question of resection of the head of the humerus and the formation of an ankylosed joint may be considered. Dislocation of the lower jaw is less common. Fractures of the patella and of some of the long bones have been described as occurring during convulsive seizures. Féré[1] describes fracture of the clavicle, humerus, the bones of the forearm and of the skull as having been seen in autopsies upon epileptics of old standing.

(*d*) *Hemorrhages and ecchymoses.* The most common site for them is the subconjunctival tissue, the whole of the sclerotic coat being sometimes obscured by the effusion. Its occurrence establishes the epileptic nature of an attack, whose true significance may otherwise be doubtful. Hemorrhages, petechial and of larger size, have been described as occurring under the skin of the face, eyelids and neck during the cyanotic stage of the fits (Spratling, Clark, Pichler, Bychowski, and others); but these must be unusual in uncomplicated cases of epilepsy.

Punctiform hemorrhages in the brains of epileptics, dying during the status epilepticus, are not uncommon, and are described on p. 178.

(*e*) The scars of *burns* and *scalds* are very common in epileptics. The resulting deformities are many and varied, and may occur in any part of the body. The disfiguration may at times be great, even to the extent of the loss of one or both eyes.

(*f*) There have been from time to time recorded a number of rarer accidents, which it is only necessary to mention: emphysema of the skin of the neck and face apparently arising from rupture of the tracheal mucosa, hernia (both inguinal and umbilical) and aural haematoma.

(*g*) Accidents of various kinds during the fit may lead to the *death* of the patient; such are, suffocation either from the passage of food into the respiratory passages, or from turning

[1] Féré, *op. cit.*, p. 429.

over on to the face in fits during sleep, or in a bath, and sudden paralysis of the respiratory centre.[1]

THE PHYSICAL CONDITION OF EPILEPTICS.

In the earlier pages of this work, attention has from time to time been drawn to the interparoxysmal state found in epilepsy, more especially with reference to the stigmata of degeneration and the permanent mental state, which are frequent and characteristic phenomena of the inherited degenerative disposition. Further reference need not therefore be made to them.

In the present section it is only intended to briefly review the general physical features of epileptics observed in the intervals of the seizures.

And in the first place, reference may be made to what has been termed the "epileptic face." According to Spratling, the *facies epileptica* results from a combination of dementia, facial scars and bromide acne. The close relation existing between facial expression and the mental state is so obvious, that it is scarcely necessary to mention the association; but on an earlier page (151) I have endeavoured to show that although the epileptic face goes *pari passu* with the degree of mental infirmity, it may also be seen in those with mental integrity, as well as in those in whom the convulsions have been arrested. Although it may be defined as an expression of dulness and heaviness, combined with emotional immobility of the features, it yet presents a stamp which seems to be peculiar to the epileptic. No doubt this appearance is augmented by the presence of the scars of bromide acne, but it may also be detected in cases presenting no such cutaneous eruption.

The epileptic may not differ in physical development from those not subject to fits, as amongst them there are found all grades from the physically robust and powerful to the frail and feeble. Many epileptic youths are undersized, which is often their only physical stigma of degeneration. As a rule they suffer from cold hands and feet, sometimes to the extent of an obvious vasomotor paralysis. The skin is frequently coarse and oily, and emits a peculiar musty odour. Their movements are usually slow, and their attitude bent and slouching. Constipation is one of their most common and troublesome

[1] Brouardel, *Death and Sudden Death*, 1902, p. 185.

symptoms. Browning[1] has called attention to the character of their bowel-discharges, which he finds made up largely of coarse unmasticated and undigested fragments of food. This is no doubt partly due to their imperfect method of mastication, as they are notoriously large and rapid eaters, and their teeth are often carious and defective.

The body weight of the epileptic does not vary much, unless in association with frequent and severe fits. Although it has been said that the immediate effect of an epileptic convulsion is to temporarily reduce the weight, there is not complete harmony amongst observers on this point. I have, however, seen the body weight fall to the extent of many pounds during the development of dementia.

Fertility in woman does not seem to be affected by epilepsy, while the influence and effect of pregnancy and child-bearing upon the disease has been already fully discussed (p. 43). Confirmed male epileptics are often given over to perverted sexual behaviour, a condition which is probably part of the co-existent mental infirmity.

Apart from the transitory convulsive symptoms of the disease, epileptics, in the interparoxysmal periods, are subject to various occasional motor disturbances. These are of several kinds, and include, those spasmodic "jerks," "jumps," or "starts," which have been already described as prodromal motor symptoms, and which are held to be instances of the minor type of convulsion. In addition *tremor* is a not uncommon interparoxysmal symptom,[2] being usually a fine rapid tremor of the neurasthenic type. Féré[3] has observed and figured tremor of the hands following the convulsive seizures, and Binswanger[4] refers to a well-marked "intention" tremor, sometimes seen in the intervals of fits.

Amongst other motor symptoms, increased myotatic irritability, and active knee jerks may be mentioned.

Articulation is often slow, and sometimes scanning; the tongue is large, atonic, frequently coated and tremulous—symptoms which may be suggestive of bromism.

The association of epilepsy with general bodily disease, and the bearing of the latter upon treatment, necessitates an examination

[1] Browning, *Journ. of Nerv. and Ment. Dis.*, 1893.

[2] R. Reynolds, *op. cit.* [3] Féré, *op. cit.*, p. 163.

[4] Binswanger, *op. cit.*, p. 307.

into the organs of special sense, such as the nose, eyes, and ears. An interparoxysmal symptom of some interest is the state of the pupils. These are sometimes found unequal on the two sides (anisocoria).

As the breath is foul, the teeth carious, and the tongue coated, a state of oral sepsis would seem to be present in the majority of confirmed epileptics, in whom also dyspepsia is not an uncommon symptom.

The connection between epilepsy and cardiac and pulmonary disease, has been the subject of considerable investigation; but from a scrutiny of the records of a large number of cases of epilepsy, in which enquiry had been made into the state of the heart and lungs, I came to the conclusion that this association was merely accidental. Of 937 cases in which the state of the heart and lungs had been noted, valvular disease of the heart was found in only 17, or 1·8 per cent., and active pulmonary tubercle in 6, or ·6 per cent.[1] Spratling's figures upon cardiac disease in epilepsy are largely in excess of those just given, viz., 238 cases out of 1070 epileptics, or 22 per cent. Even eliminating a number of cases in which the cardiac disturbance appeared to be of a functional character, the percentage of the organic valvular disorders worked out at 15 per cent.

In like manner I did not find pulmonary tubercle a frequent complication of epilepsy, although, according to Spratling, 25 per cent. of his cases of confirmed epilepsy died of this disease.

[1] These figures are not to be considered final on this point, as the relation between epilepsy and heart disease is under investigation.

CHAPTER VIII.

THE PATHOLOGY OF EPILEPSY.

Pathological anatomy—Changes pointing to defective development—Acute change with swollen nucleus—Chronic change with shrinking and dark staining of cell and nucleus—Changes resulting from epileptic seizures—Neurogliosis—Incidence of gliosis—Sclerosis of cornu ammonis—Cortical haemorrhages—Changes in thalamus, blood-vessels (angeiomata), cerebellum, medulla oblongata, spinal cord (tract degenerations)—Albuminous exudate in peri-vascular spaces—Changes which appear to act as causal factors of attacks—Thrombi, various forms of—Calcareous or vitreous deposits in the cortex, etc.—Summary of pathological appearances.

THE PATHOLOGICAL ANATOMY OF EPILEPSY.

(For this chapter on the pathological anatomy of epilepsy I am indebted to my friend Dr. John Turner, one of the medical officers of the Essex County Asylum.)

It is not surprising that a disorder with such striking clinical characteristics as epilepsy should have engaged the attention of histologists ever since the dawn of neuro-histology. Almost all observers have noted an increase of the neuro-glia elements, engorgement of the blood-vessels, diminution in the number and changes in the structure of the ganglion cells, and atrophy and sclerosis of the cerebral convolutions; while some have detected changes in the structure of individual nerve cells, such as the vacuolation of the nucleus described by Bevan Lewis, and the enlarged globose cell described by Mott in conditions of status epilepticus. It is now generally admitted that, with the exception of the two last described, these changes are the result of a morbid condition which is associated with, or set up as a result of, epileptic fits, and cannot be regarded as anatomical causes of the seizures.

The writer has, like most other observers, based his opinions largely on changes met with in the brains of congenitally

defective individuals. The great preponderance of epilepsy among this class points to the conclusion that the structure of their brains offers great facilities for the development of this disease. So vastly more frequent is epilepsy among the congenitally deficient, that it may almost be looked upon as a stigma of degeneration. The occurrence of epilepsy in sane persons is not necessarily an objection to this view, as congenital structural defect may co-exist with average intelligence.

Before passing to a detailed discussion of the changes found in the central nervous system of epileptics, it will perhaps enable the reader to better appreciate the significance, which the writer attaches to them, if a brief account is given of his views concerning what he regards as the immediate cause of epileptic convulsions. He believes that epilepsy is a disease occurring in persons with a defectively developed nervous system, indicated by certain structural peculiarities, and in whom there is a special tendency to intra-vascular clotting; that the convulsions are a symptom of the disease, and that prior to a seizure a condition of cortical stasis is induced by the formation of intra-vascular coagula, which become lodged in some of the cortical blood-vessels; and that in the process of coagulation the blood plates play a prominent part. It is also contended that intra-vascular coagulation will explain both the atrophy and sclerosis, and the blood tumours found in many epileptic brains. To account for the former the blockage is believed to take place on the arterial side, leading to starvation of the cortical tissues; while the latter is explained by a blocking of the veins so as to cause a local rise in the arterial pressure, which, after the lapse of time, results in enormous dilatation of the capillaries. The microscopical appearances of the blood tumours, and the frequent occurrence of haemorrhage within them, favour this supposition.

The view that epileptic fits may be caused by plugging of small cerebral arteries is not new, as it was advocated by Hughlings Jackson[1] in 1864. His hypothesis, however, did not include what is here believed to be an essential part of the process—a congenital structural defect of the nerve cells. It is true that he referred to an over-irritable condition of these cells, a result of malnutrition from the plugging of the vessels, but not to a congenital hereditary deficiency.

[1] Jackson, Hughlings, *London Hosp. Reports*, 1864, i.; *Brit. Med. Jour.*, 1888, ii.

In reviewing the various lesions which have been described, it will be found convenient to divide them into:

(*a*) Those which are accompaniments, or stigmata, of a defectively developed nervous system.

(*b*) Those which are probably the results of epileptic convulsions.

(*c*) Those which, according to the writer, appear to act as causal factors in the production of the attacks.

(*a*) Changes in the nervous elements of the cerebral cortex mainly indicative of a congenitally defective nervous system—cerebral stigmata.

Tangential fibres. No marked changes were found in these fibres in the few cases examined for the purpose; but Kaes[1] has described in three instances a peculiar disposition of the association fibres immediately subjacent to the zonal layer, whereby a fibreless space occurs in the inner half of the first layer, under which lies a smaller parallel stripe of closely set, often varicose, fibres. This is best observed in the occipital region, but is met with also in the frontal and central regions.

Nerve cells. The nerve cells are diminished in number, approximately to the degree of imbecility. This diminution affects chiefly the cells lying in the outer half of the cortex and seems fairly universal in all the regions examined, except the occipital, where it is less evident. In some cases the reduction in number is most marked in the second layer, in others in the smaller pyramids of the third layer. The individual cells are often stunted and with few processes; but only in very low-grade imbeciles or idiots—according to the experience of the writer—is it usual to meet with the rounded or pyriform cells described as characteristic of imbecility. In almost all cases individual cells and groups of cells are manifestly shrunken, their cytoplasm staining darkly, their nucleus small, dense, or homogeneous. Such cells are without doubt degenerated, and in advanced stages lose all affinity for basic dyes.

Bevan Lewis[2] attached much importance to the occurrence of vacuolation of the nucleus, especially when it affected the cells of the second cortical layer. This is a condition which only reveals itself in sections prepared according to that author's

[1] Kaes, *Neurol. Centralblatt*, 1904.

[2] Lewis, Bevan, *Text Book of Mental Diseases*, London, 1889.

method, which has not been made use of in this investigation. Campbell[1] has shown, however, that it is not uncommonly met with in various other diseases, such as tubercle and pneumonia, and that it has no definite relation to the epileptic condition.

Andriezen[2] referred to a thickening of the intra-nuclear reticulum and to a fine dust-like material in the nucleus of the nerve cells in epileptics. Quite recently Clark and Prout[3] have laid stress on the early occurrence of changes in the nucleus. They find disappearance of the nuclear membrane and network, so that the interior of the nucleus is not reticular, but occupied by granules, in consequence of which the nucleolus, no longer held in place by the network, is apt to be displaced. These observations only in part coincide with those made by the writer. In many cases the nucleus, especially of the second layer and spindle layer cells, is large, clear, and "bladder-like," and often bulges out beyond the normal contour of the cell body, giving to the cells an appearance extremely like that produced in the dog's brain after ligation of the cerebral arteries (Fig. 3); but one often finds associated in the same section cells whose nuclei are dark and dense, or even quite homogeneous; or sometimes, whilst one region of the cortex shows a preponderance of cells with large clear nuclei, another region shows a majority of cells with small dense nuclei. These two conditions represent probably an acute and a chronic change, respectively; the swollen nuclei ultimately becoming shrunken and darkly stained. There is no disappearance of the nuclear membrane, but full endorsement can be given to the statement of Clark and Prout concerning the disappearance of the network structure and its replacement by granules which stain red with erythrosin, an appearance very commonly seen in other brains than those of epileptics.

The only change calling for special remark concerning the nucleolus is that it is not at all infrequently enlarged, measuring for example in a Betz cell as much as seven to nine microns instead of four or five; but this condition is more strikingly shown when film preparations of cells are made, when it sometimes appears as two or three times its normal diameter. Usually in this enlarged state it stains less darkly, or may have a pale centre and a dark rim.

[1] Campbell, A. W., *Jour. of Path. and Bact.*, ii., 1894.

[2] Andriezen, W. L., *Brit. Med. Jour.*, i., 1897.

[3] Clark and Prout, *Am. Jour. of Insanity*, 1903–4.

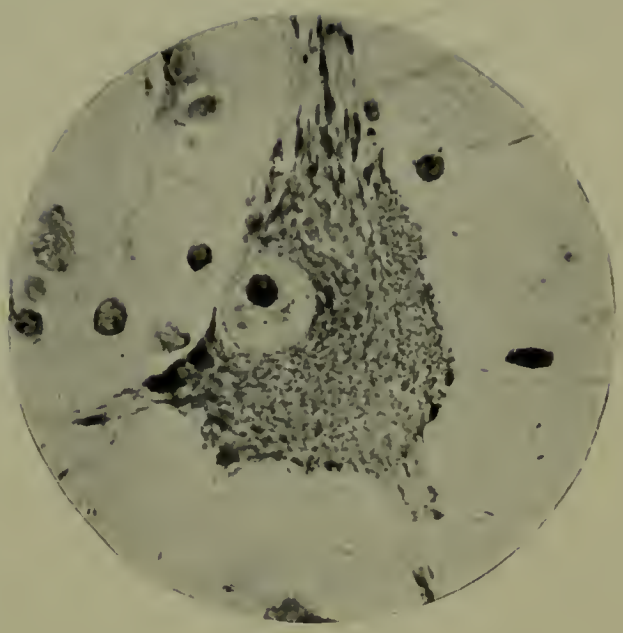


FIG. 1. ×600.

Betz cell from an epileptic imbecile, showing the characters described in the text which indicate defective development.

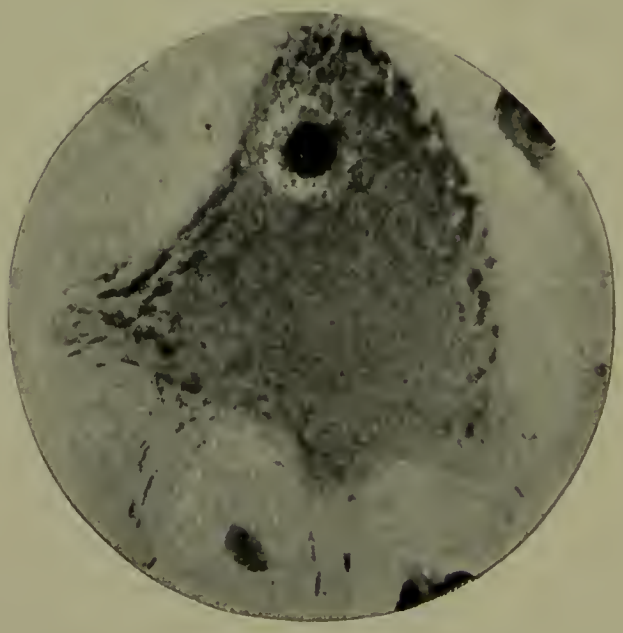


FIG. 2. ×600.

A similar form of cell from another case of epilepsy with enlarged nucleolus.

Facing page 169.

Mott[1] has described in conditions of status epilepticus a swollen dropsical appearance of the cells, with diffuse chromatolysis and an enlarged eccentric nucleus. From the figure he gives it would seem as if he had taken, as characteristic of status, a form of cell which is found in the great majority of imbeciles, whether epileptic or not. It is most marked in the large Betz cells, and bears a resemblance to the *réaction à distance* type (Figs. 1 and 2), which is set up after division of motor axons; the cell body is large, and the chromophilic flakes well marked at its periphery and in its main branches, but its centre is occupied by fine closely-set granules. The nucleus, which is generally large, plump, and clear, is displaced, and situated quite to one side, or right up in the apical portion. This is an embryonic type, and, as Lugaro[2] has pointed out, is normal in some of the nerve cells of the lower vertebrates. The cells of the hypoglossal nucleus and anterior horns of the cord do not usually show the change except in idiots and low-grade imbeciles. This axonal form of cell was found in 27 out of 35 cases examined.

Probably also the "marked chromatolysis, often general, but sometimes central," which Clark and Prout[3] find so common, especially in the third layer, is merely a feature of the nerve cells in imbeciles, and has no special relation to the epileptic condition.

In the white matter and in the outer layer of the cortex of the brains of the lower vertebrates at all periods of life, in new-born infants and in imbeciles at all ages, there are numbers of nerve cells, but in normal adults these cells have almost entirely disappeared from the outer layer, and to a large extent from the white matter. Roncoroni[4] was the first to point to this persistence of nerve cells in the white matter as an embryonic type.

Clark and Prout have stated their view that the cells most affected in epilepsy are those of the second and third cortical layers, from which, in accordance with the view widely maintained, that the second layer cells are of a sensory nature, they infer that epilepsy is a sensory phenomenon. But the sensory function of the second layer is by no means proven;

[1] Mott, F. W., *Croonian Lectures*, 1900, London.

[2] Lugaro, E., *Riv. Sperimental di Fren.*, 1902.

[3] Clark and Prout, *Am. Jour. of Insanity*, 1900.

[4] Roncoroni, *Arch. di Psich.*, 1896.

indeed, the most recent histological investigations of Campbell[1] seem to place the cortical centre for this function in the post-central gyrus.

The great prevalence of epilepsy amongst congenitally defective persons is a sign that their imperfectly developed nervous system is in a morbidly unstable state, and in consequence apt to be affected by circumstances which in normally developed and stable brains would produce no marked results.

(*b*) Changes resulting from epileptic attacks.

Membranes. The brain of an epileptic is usually bulky, its convolutions simple, and its membranes to the naked eye clear and not apparently thickened; they are not adherent to the cortex, and are congested only in cases which die in the status epilepticus. Microscopical examination shows, however, that there is usually some fibroid thickening. In uncomplicated cases there is very little cellular infiltration; what there is consists of lymphocytes, for the most part of the large variety. Plasma and "mast" cells are absent both here and in the neighbourhood of the cortical vessels; a solitary instance of each occurred in two cases. Although to the naked eye there may be no signs of congestion, it will be found that the veins are engorged, and in every case examined there was observed a more or less extensive extravasation of red corpuscles, resulting evidently from ruptured blood-vessels. Sometimes the muscular coat of the arteries is thickened, and in acquired epilepsy, especially the senile forms, endarteritis is very common, even leading to the complete obliteration of the vessel's lumen. Besides the red cells it is common to see in the vessels masses of fibrin threads. These deposits are met with in cases uncomplicated with any fibrinous inflammation of the thoracic or abdominal viscera, although when such is present the threads of fibrin are more than ordinarily plentiful. Clusters of leucocytes are frequently seen lying among the meshes of the fibrin threads, or they may enclose a number of red blood corpuscles. Other deposits are also met with, taking the form of spheres, which may exist separately, or be clustered together into little mulberry-like masses, which will be dealt with more fully when describing the cortical blood-vessels. In

[1] Campbell, A. W., *Histological Studies on the Localisation of Cerebral Function*, Cambridge, 1905.

some cases amyloid bodies lie in the superficial layers of the pia mater.

There is one feature constantly present and highly characteristic of epilepsy, although sometimes found in small amount in other diseases (notably general paralysis), viz.: a foam-like or frothy exudate lying between the membranes and the surface of the cortex. It contains no nuclei, and is obviously an unorganised structure representing, probably, the coagulation of an albuminous exudation from the vessels. It occurs also in large amount in the peri-vascular spaces, and was a feature on which Schröder van der Kolk[1] laid great stress. It is probable that by obstructing the lymph spaces it may interfere with the nourishment of the adjacent tissues, and the fatty changes which it undergoes may still further impair metabolism by setting free decomposition products which might act as direct poisons to the nerve cells.

Neuroglia—Neurogliosis. In recent years great importance has been attached to neurogliosis, some authorities even going so far as to regard it as the cause, or one of the causes, of epileptic fits. The region most prone to undergo increase in its glia cells and fibres is undoubtedly the first or outer layer of the cortex; in many cases this is the only region affected, but in others there may be a marked increase throughout the whole cortex. Next to the outer layer, the white matter in the regions adjoining the cortex is that most often affected. The first or outer layer of the cortex appears unduly deep, owing probably to the very marked diminution of the cells which form the second layer. There is in many cases a patchy sclerosis of the surface, consisting of fibres which lie closely packed together, and running for the most part in a direction parallel to the surface of the convolution. This rim may in some places reach a thickness of over a hundred microns, although it usually does not average more than 30 or 40. At the junction of the sclerosed rim with the cortex, glia nuclei lie closely together, but very few are met with actually within the sclerosed portion. The glia cells of this layer are generally increased in number, more especially in the deeper parts. Not infrequently definite little buds, formed by a localised neuroglial exuberance, appear as outgrowths from the surface. A careful search usually reveals embryonic nerve cells.

[1] Kolk, Schröder van der, *New Syd. Soc. Trans.*, 1859, London.

Although the majority of the glia cells are of small size, occasionally gigantic forms are seen, giving to the part where they occur a leather-like consistency, much above that of the brain in general. Sometimes these big cells contain several nuclei.

In the brains which formed the material for this chapter, gliosis was not a common feature, and in those in which it was present, was not more marked than is commonly seen in any chronic forms of insanity.

Its incidence was as follows:

In ten cases where the frontal convolutions were examined, a gliosis of the first layer was present in three, and in none was it marked.

In eight out of twenty-seven cases there was some gliosis of the first layer of the ascending frontal gyrus, with a superficial sclerosed rim, varying in depth from 30 to 130 microns.

In six cases the region of the calcarine fissure failed to show any gliosis.

In eight out of thirteen cases there was evidence of gliosis of the cornu Ammonis, affecting not only the surface, but the whole thickness of this part.

In two out of six cases granulations were found on the floor of the fourth ventricle.

In three out of eleven cases the Bergmann's fibres of the cerebellum were marked.

Bleuler[1] found in the brains of twenty-six epilepties a definite hypertrophy of the bundles of neuroglia lying between the pia mater and the outermost nerve bundles. He describes this condition as having the appearance of a scar more or less detached from the subjacent tissue, and occurring over the whole brain; and he concludes that diffuse subpial gliosis is a constantly present condition in epileptic dementia. This statement appears to need some qualification, for, although a superficial stratum of glial tissue is met with in epilepties, it is generally very localised, so that whilst one part of the surface of a convolution may show it, it may be quite absent from neighbouring parts. When present it varies in thickness from 20 to 30 microns (at which degree it is by no means peculiar to epilepties' brains) up to perhaps several hundred. As was noted when dealing with the outer layer, the increase

[1] Bleuler, *Münch. Med. Woch.*, 1895.

of glial tissue sometimes takes the form of little excrescences, or buds, similar to those seen on the fourth or lateral ventricles in general paralysis. Clark and Pront, who have also drawn attention to this local overgrowth, figure one of these. They also refer to the varied condition of the glial nuclei, some being small, dense, and irregular; others larger, clearer, and with definite chromatin granules; while others are very large indeed. It is not at all uncommon to find also on the floor of the fourth ventricle these little neuroglial buds, but for the most part they occur singly or in small groups, and are situated more at the lateral borders of the floor, away from the central line, a condition the reverse to that which is found in general paralysis.

Sclerosis of the cornu Ammonis. For more than half a century this feature has attracted the attention of pathologists. By some it has been credited with great significance; by others it has been passed over as of minor importance, and at the most as only a secondary manifestation. Facts are still lacking which would enable us to definitely decide between these views, and by the mere examination of morbid tissues, unaided by experimental research, the histologist can scarcely expect ever to be in a position to do so. Nevertheless, histology is of considerable service, and furnishes valuable hints towards a solution of the problem.

The latest writers appear to minimise its importance, or not even to allude to it at all; but before proceeding to personal observations, a short account will be given of the results arrived at by some previous workers.

Jelgersma[1] refers to sclerosis of one or both of the horns as a change often associated with epilepsy. He found it most frequently in chronic cases and does not think that there are sufficient grounds for regarding it as a causal influence. It occurred in about twenty-four per cent. of his cases. He draws attention to what he considers an important fact—that it may be merely the part of a sclerosis affecting nearly the whole cerebrum. He concludes that epilepsy is most likely the cause of the change, and mentions that it is found (but rarely) in other cases.

Fischer[2] from the examination of two cases, in both of

[1] Jelgersma, *Nederland. Tijdsch. v. Geneeskunde*, 1888.

[2] Fischer, *Neurol. Centralblatt*, 1893.

which one horn was more affected than the other, cites them in support of Wundt's view that atrophy and hardening are dependent upon asymmetrical enlargement of the lateral ventricles, which enlargement Fischer supposes to result from the disturbance of the circulation accompanying all epileptic fits.

Worcester[1] states that the preponderance of authority favours the view that this lesion is the result, rather than the cause, of epilepsy. In forty-three cases examined, he found it on one or both sides in twenty: in eleven of which no other abnormality was observed; and in nine it was accompanied by other and more extensive changes, which he believes have a common origin with it. The histological characters of this condition he found to be remarkably uniform, chiefly a general sclerosis involving destruction of the neurones having their origin in the stratum pyramidale and nucleus fasciae dentatae. He failed to note this condition in over one hundred and fifty brains from other forms of insanity, except in one general paralytic. He favours the idea that sclerosis of Ammon's horn is the cause of the convulsions, and argues in support of this view that a cicatrix of the cortex may act as a focus of irritation, whether situated in the temporo-sphenoidal lobes or in any other part of the cerebral cortex.

Ford Robertson[2] states that he met with it in one out of thirteen cases, but does not appear to attach much significance to it. Binswanger and Oppenheim, Bratz[3] and Weber[4] all look upon it as a developmental defect. Weber, however, records a case in which it was present, where the epilepsy had supervened late in life after chronic lead poisoning.

Personal observations. In 115 cases of epilepsy the writer found the cornu Ammonis sclerosed on one or both sides in nearly half: in ten cases on the right side only, in twenty on the left, and in twenty-four on both sides. The sclerosis, however, seems to have a greater tendency to affect the left side than these figures show, for when both sides were affected the sclerosis was generally more marked on the left. Of the 115 cases, sixty-two were men, amongst whom it occurred twenty-six

[1] Worcester, W. L., *Jour. of Nerv. and Ment. Dis.*, 1897.

[2] Robertson, Ford, *Pathology of Mental Disease*, Edinburgh, 1900.

[3] Bratz, *Arch. f. Psych.*, xxx., 1, 3.

[4] Weber, L. W., *Beit. z. Path. u. Pathol. Anat. d. Epilepsie*, 1901.

times; fifty-three were women, in whom it occurred twenty-eight times. The opinion of Jelgersma and others, that it is only a local manifestation of a more general propensity towards atrophy, is probably correct, for it is commonly associated with atrophy in other regions, notably the occipital lobes and the cerebellum; and sometimes indeed when the horns were unaffected, an atrophic condition existed elsewhere. So that if the latter cases were also included, *atrophy, and sclerosis, of some part of the central nervous system, were found in very considerably more than half the cases of idiopathic epilepsy.*

Sclerosis of the cornu Ammonis appears to be an extremely rare circumstance as an isolated feature in other than epileptic brains. There are cases of general paralysis where the entire brain is atrophied and sclerosed, and in these the cornu Ammonis is naturally involved. The condition which is being discussed, however, refers to one that implicates the cornu Ammonis in a localised way, so that its consistency is much harder than that of the surrounding cortex.

In addition to general paralysis, the writer has only found it in three cases, which were not epileptic. One of them had been diagnosed as general paralysis, a view which was not confirmed at the post-mortem examination, nor by the microscopical investigation of the brain, and it is quite possible that this case (which was only under observation a short time) might have been an epileptic. Of the other two, one was an imbecile, and the third was a case of senile dementia.

As a rule, the sclerosed condition of the cornu Ammonis is not due to any manifest overgrowth of the glia elements, but rather to a shrinking of the tissues generally, and disappearance of the nervous elements, whereby the neuroglia fibrils become more closely approximated and more conspicuous. There are several facts which lead one to suggest that the changes are due to a deprivation of the normal blood supply, in consequence of which the nervous elements become starved and shrunken. This condition is evidently allied to the atrophied areas met with in the convolutions of other parts, especially in the occipital region, and the extremely common atrophied foliae of the cerebellum, which are often not discoverable on a naked-eye inspection. The sharply defined, localised nature of the lesion conforms with the idea that it is due to interference with the vascular supply.

In transverse sections of the selerosed horns there is very little beyond the manifest atrophy and diminution in the number of the nerve eells. It is by comparing sections from the two sides of the brain in cases where only one horn is sclerosed that a true idea is obtained of the great number of nerve cells, whieh have disappeared on the affected side, while at the same time the shrunken condition of those still present is also more readily appreciated. There is generally a more or less pronounced sclerosed rim along the surface of that part of the horn which lies free in the ventricle, and this may be raised up into little excreseences forming a granular surface; but where such is the case they are seen on both sides even when only one horn is manifestly atrophied, showing that this rim is not a feature specially related to the atrophy, but is part of a general selerosis.

In a few cases, more especially when the sclerosis and shrinking is not marked, an increase in the glia elements occurs, and numerous large pale glia cells are seen lying between the nerve eells.

The histological characters are, as Worcester observes, remarkably uniform. In the writer's opinion this lesion is not only extremely characteristic of idiopathic epilepsy, but, from the manner in which he believes it to originate, appears to be very closely associated with the conditions which determine the fits.

It was evident that in the great majority of cases there were no appearanees of any active pathologieal process. There was no exeessive overgrowth of the glia elements, but merely a disappearanee of nerve cells, and a general shrinking, due, it is suggested, to a starvation of the tissues by deficiency of their blood supply. This deficiency is aceounted for by the partial or eomplete ocelusion of the nutrient vessels by intra-vascular clots. The nutrition of the part is probably still further interfered with by the presence of albuminous exudate found in the lymph spaees.

Although it is fully recognised that sudden general anaemia of the brain is followed by eonvulsions, it is not known whether a fit will result from the sudden deprivation of the blood supply to small areas of the eortex. But granting for the moment that this may oceur, the fits would only last until the nerve eells implicated beeame exhausted; the cause of the

block still remaining, a secondary result would be a shrinking and hardening of the affected parts. If, however, the vessels were not completely occluded, the effect would be merely to diminish the blood supply, which in itself would tend to the devitalising and atrophy of the parts thus deprived of their normal amount of blood.

This occlusion of the nutrient vessel, as a factor in the production of sclerosis of the cornu Ammonis, is no mere hypothetical condition, but one which can be demonstrated.[1]

It is somewhat difficult to suggest an entirely satisfactory reason why the sclerosis should so often select this part in preference to others. That in some way it is connected with the blood supply appears highly probable, for, as Worcester says, we have no reason to suppose that lesions of this part exercise any special prerogative in exciting convulsions.

The small cortical haemorrhages found in the brain of a rabbit, which had died in convulsions after the intravenous injection of clove oil, were more numerous in the olfactory lobes and those parts of the cortex in the vicinity of the olfactory nerves than elsewhere: an observation which would seem to strengthen the view that there is some close connection between the site of the lesion and the disposition of the blood-vessels, for in all probability these small haemorrhages are due to the impaction of emboli carried by the blood stream to the small vessels of these parts, and leading to their rupture. So also the greater frequency of the lesion on the left side may be due to the arrangement of the nutrient vessels, for whilst, on the right side, the carotid is given off from the innominate artery, on the left it springs directly from the aorta. To this disposition of the vessels has long been assigned the reason for the greater frequency of gross cerebral embolism on the left side.

Wundt's hypothesis, referred to by Fischer,[2] that the greater frequency of the lesion on one side depends upon asymmetrical enlargement of the lateral ventricles, will not bear investigation. The only direct effect of such a condition on the cornu Ammonis would arise from the presence of the

[1] Recently, in one of the rare cases where sclerosis of the cornu Ammonis occurred in a non-epileptic, the posterior cerebral artery on both sides was found to be the seat of extensive endarteritis and occlusion of its lumen.

[2] Fischer, *Neurol. Centralblatt*, 1893.

fluid contents, which freely communicate between the two ventricles, and how this can affect one side only it is difficult to see. He has apparently confused cause and effect, for obviously an atrophied horn will be associated with the larger ventricle, of which it helps to form a boundary.

Optic thalamus. B. Onuf[1] has drawn attention to an atrophied condition of the optic thalamus in epileptics. In nine cases which he examined it was found atrophied in seven: three times on the left side, once on the right, and twice on both sides. This atrophy may be accounted for in the same way as that of the cornu Ammonis, and it is interesting to find that as with the latter, so with the optic thalamus, the left side shows the greater liability to suffer. In three cases examined by the writer there was found a very slight shrinking of the thalmus in all: once on the right side and twice on the left.

Blood-vessels. In the great majority of cases the walls of the blood-vessels show no structural alteration beyond perhaps a slight increase of the tunica muscularis of the arteries. Congestion has been noted by every observer, and it is a prominent and constant feature, most marked in those cases which have died in status epilepticus. To be more precise, it is the veins and capillaries which are engorged, the arterioles in most instances being empty, and in consequence showing a collapsed appearance. In longitudinal sections the vessels generally appear more or less tortuous, and owing to their empty condition the peri-vascular lymph spaces appear dilated. Probably this collapsed condition is to some extent due to contraction of the arterial walls at the time of death. Correlated with this congested state of the veins are numerous small haemorrhages, which occur in meninges, cortex, pons Varolii, cerebellum, and generally throughout the brain. Meningeal haemorrhages are found even in cases which to the naked eye showed thin, clear membranes, and an anaemic aspect of the cortex, and they have been a constant feature in all the epileptic brains examined.

In sections the number of white corpuscles seen in the vessels is not, as a rule, large, although blood film preparations usually reveal a considerable leucocytosis. The vessels lie in dilated lymph spaces, which do not contain many proliferated nuclei,

[1] Onuf, B., *Jour. Am. Med. Assoc.*, April, 1905.

but sometimes here and there are collections of lymphocytes, generally of the large variety, and the foam-like exudate is constant. Around the capillaries, it is true, there is often an increase of nuclei, which, however, appear to belong to the endothelial cells, lining these channels, and which have undergone some proliferation. An important point which differentiates them from the large lymphocytes, is their adhesion to the side of the lymph channel, away from the capillary wall, whereas proliferated leucocytes cling closely to the latter part. Histologically they can be differentiated from the nuclei of the small lymphocytes by their larger size and greater clearness, and from those of the larger lymphocytes by the absence of any attached protoplasm, which the latter always show when stained in a similar way. Weber,[1] who denies the existence of an endothelial lining to the lymph spaces, attributes these bodies to an increase of the glia elements, which according to him take the place of an endothelial lining.

Angeiomata or blood tumours. Among the changes in the blood-vessels must be mentioned the presence of angeiomata or blood tumours, which, although not peculiar to epileptics, appear to be specially liable to occur in them. They are probably much more common than is usually considered, but their frequent small size allows them to be easily overlooked, more especially as they may be located in the spinal cord, a region which is frequently not examined in autopsies. They consisted in all cases of dilated blood-vessels, chiefly capillaries, with more or less intervening brain substance, in which were large spider cells. The walls of the capillaries were sometimes of extreme thinness, and not unfrequently had ruptured. Drysdale[2] has reported two cases, both in epileptics. Creite[3] reports one in the meninges of an epileptic. The view most generally accepted as to the cause of these angeiomata is that suggested by Virchow, that they are of congenital origin. It is probable that a congenital defect is a predisposing factor in their production, but the apparent rarity of the condition in imbeciles, not affected by epileptic fits, points to the presence of some other factor, and this, it is suggested, is rise of blood pressure due to an obstruction of the blood stream on the

[1] Weber, L. W., *Beit. z. Path. u. Pathol. Anat. d. Epilepsie*, 1901.

[2] Drysdale, J. H., *Trans. Path. Soc., London*, 1904.

[3] Creite, *Münch. Med. Woch.*, 1903.

venous side of the circulation, leading to enormous dilatation of the capillaries, and thickening and dilatation of the arteries. In all of the cases which were specially examined on the point, abundant evidence of intra-vascular coagulation was found.

Cerebellum. It is a well-recognised fact that in epileptics the cerebellum is small in proportion to the remainder of the brain, but apart from this developmental defect local atrophy of the foliae is a common feature.

The atrophied patches may be so small as to be only discovered on microscopical inspection. The lesions found in this part of the brain are similar to those occurring in the cerebrum, and are nearly, or quite, as well marked. The membranes have undergone some fibroid thickening, and are infiltrated to a moderate extent with large lymphocytes. Haemorrhages are invariably present, and often more marked than in the cerebral membranes. The delicate foam-like exudate is copious, and seen in all cases. As a rule, evidences of glial overgrowth are not conspicuous. The cells of Purkinjé appear to be diminished in number. The majority are pale, and show well-marked central chromatolysis. The main branches are inconspicuous, owing to absence of Nissl bodies. Shrunken and densely stained forms, either singly or in groups, are also found in most sections.

There appears also to be in some cases, apart from the obviously atrophied regions, a general diminution in the number of granules. In the atrophied foliae, according to the extent of the lesion, there are few or none of either Purkinjé cells or granules.

Evidences of intra-vascular coagulation (Fig. 6) are abundant, and, although so far no actual demonstration of rupture due to blocked circulation has been met with here, the often limited, and always sharply defined, area of atrophy is strongly in favour of the lesion being due to deprivation of the blood supply.

Medulla oblongata. This was the favourite site for the localisation of the essential lesions of epilepsy by the older pathologists, and they were able to demonstrate here many fairly constant and well-marked changes, such as sclerosis and constant engorgement of the vessels, with not infrequently punctate haemorrhages. Schröder van der Kolk[1] accounted for the sclerosis by an exudation of albumen from the distended vessels, the exudation subsequently undergoing fatty degenera-

[1] Kolk, Schröder van der, *New Syd. Soc. Trans.*, 1859, London.

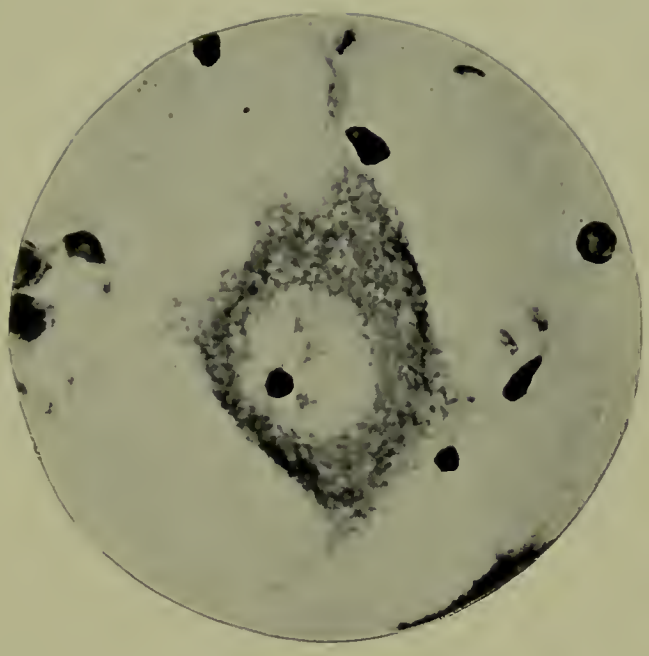


FIG. 3. ×600.

Betz cell from a case of epileptic idiocy, with enormously swollen 'bladder-like' nucleus. This form, which is commonly met with in epileptics, bears a great resemblance to the cortical nerve cells of a dog after ligature of its cerebral vessels.

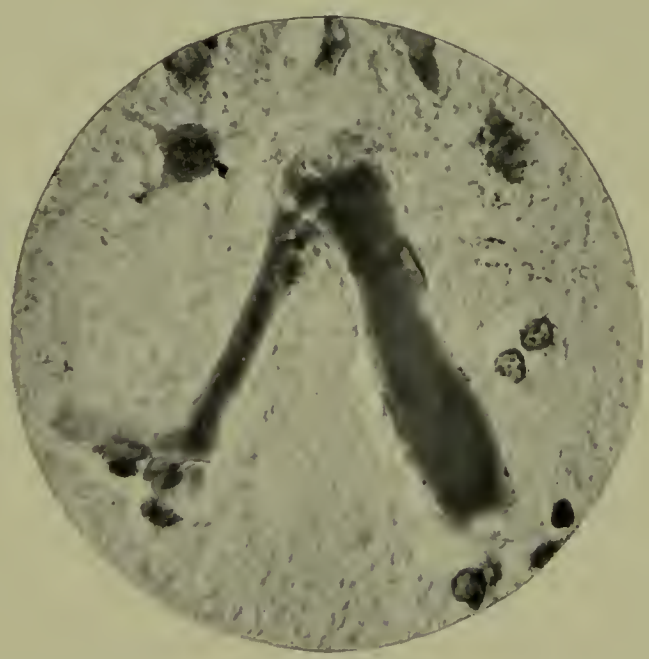


FIG. 4. ×600.

Capillaries blocked by hyaline clot. The bulging of the longer capillary is evidence of its being actively distended by the clot. From the cortex of an epileptic imbecile who died in a fit.

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tion. It is not without interest to note that there is evidence that this albuminous exudate does undergo fatty degeneration, for in Marchi preparations, besides the usual foam-like exudate in the lymph spaces, oval bodies with black borders are met with, looking like partially deflated bladders. Although mentioned specially here, where they occur in unusual numbers, they can also be seen in the lymph spaces of the cerebral cortex and of the spinal cord.

It should be noted that the large cells of the hypoglossal nucleus are not wont to show the axonal appearance, which is so common amongst the Betz cells. They are usually shrunken, lie in dilated spaces, and although denser than normal, show a stichochrome structure. The cells of both the cuneate and gracile nuclei, as in nearly every case of insanity, have an axonal appearance.

The comparatively frequent presence of small granulations on the floor of the fourth ventricle, and their peculiarities of position, have been previously noted. In histological characters they are precisely similar to those seen in general paralysis.

As Schröder van der Kolk long ago noticed, the olivary bodies are often sclerosed, and the microscopical appearances are similar to those found in the cornu Ammonis. The nerve cells are shrunken and heavily pigmented.

Spinal cord. The condition of the fore-horn cells is rarely axonal, except only in low grade idiots and imbeciles. They are usually shrunken and lie in wide spaces, but present a fairly regular disposition of their chromatoplasm; an excess of yellow pigment is very common. The nucleus is generally smaller and denser than normal, and the nucleolus may be enlarged.

The central canal may be obliterated in parts or along its whole course by a proliferation of the lining cells. When the obliteration is only local, it is usually the lower regions that suffer.

The appearance of the meninges and vessels, and the contents of the latter, are precisely similar to that which has been previously described in the brain. Small meningeal haemorrhages are common, and at times punctate haemorrhages are met with in the grey matter of the horns.

Tract degenerations in the spinal cord. Considering the frequency with which degenerated cells occur in the cortex, it is not a matter for surprise that a secondary degeneration of

the pyramidal tracts is often found; this may represent bygone or recent changes.

The number of spinal cords examined for these degenerations was sixteen, and five showed no degeneration. In seven there was more or less marked degeneration of the crossed pyramidal tracts. In one case this was associated with a tumour in the right frontal region, and occurred only in the left crossed pyramidal tract.

If one may draw a conclusion from such a small number of cases, degeneration or deficiency of the fibres in the posterior columns is still more frequent, having been met with in eight cases.

(*c*) Changes which appear to act as causal factors of epileptic attacks.

The pathological changes, already described, are regarded by the writer as phenomena, which merely precede or follow the course of epileptic attacks. In the intra-vascular thrombi, about to be described, he believes, are to be found the primary cause of epileptic convulsions.

Intra-vascular clotting is found in four forms:

(*a*) Spheres or mulberry-like masses lying free in the blood-vessels (Figs. 5 and 6).

They are probably formed by an amalgamation of the blood plates, for they both contain phosphorus, and their behaviour to reagents is similar. These coagula may completely block a small blood-vessel, and even cause rupture of its wall.

(*b*) Hyaline material clinging to the vessel wall and blocking its lumen. The casts so formed appear to arise from an aggregation and fusion of the spheres just described (Fig. 4).

(*c*) Finely granulated débris, which probably represents the disintegration of the afore-mentioned casts.

(*d*) Fibrin threads. It is under this form that intra-vascular coagulation most widely occurs, but it is the form least characteristic of epilepsy. In all diseases attended by fibrinous inflammation it may be observed in the vessels, not only of the central nervous system, but of all parts of the body. The inflammatory process, when it is associated with epilepsy, is, however, generally characterised by a much more abundant deposition of fibrin than when it occurs in cases not suffering from epilepsy, and may lead to thrombosis of the large veins of the meninges,

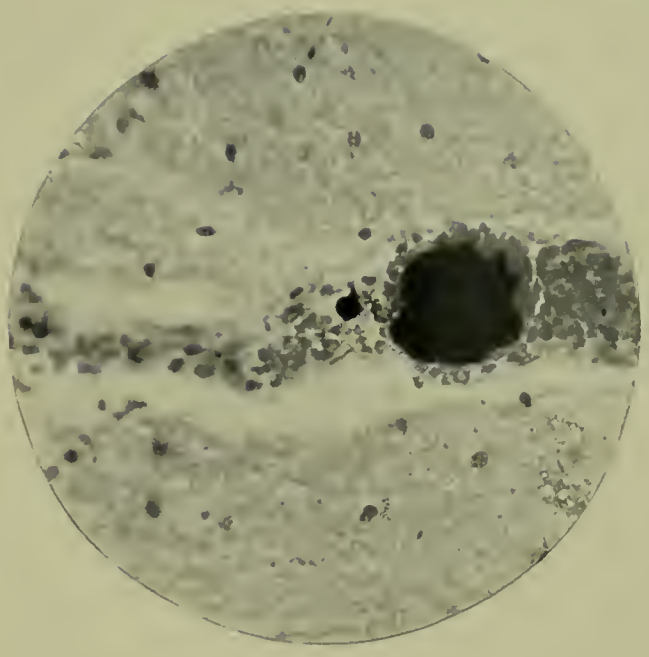


FIG. 5. ×300.

Lobulated thrombus lodged in a vessel of the Cornu Ammonis. To the left of the figure the vessel is collapsed and has ruptured, and erythrocytes are seen lying in the lymph space. Notice an isolated, darkly stained sphere near the larger mass, which latter appears to be formed by the massing together of a number of such spheres.

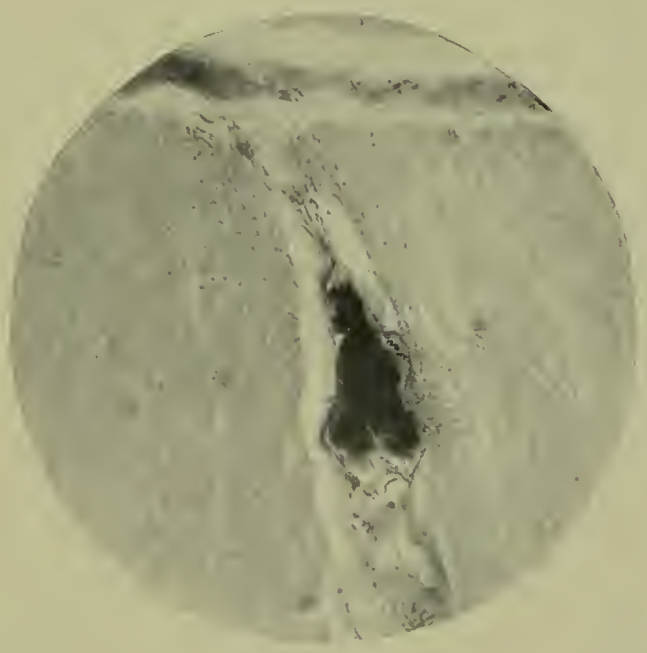


FIG. 6. ×400.

A cerebellar cortical vein in which is impacted a lobulated thrombus. Notice the collapsed condition of the vessel in its upper portion. This picture was obtained from a preparation stained by Macallum's method for the detection of phosphorus in tissues, and the clot was of a bright emerald green colour, showing that it contained phosphorus.

Facing page 182.

or even of the sinuses, with multiform punctate haemorrhages of the cortex. The following case illustrates this: The patient, aged 19, a low-grade imbecile subject to epilepsy, died of purulent pleurisy. Just before death she had a large number of severe fits. Blood was effused under the dura of both sides, and on the right there was a firm white thrombus in a large vein lying over the centre of the second frontal convolution. A part of the cortex of the Island of Reil was softened, and here there was an extensive blocking of the veins and capillaries with fibrin threads, which in the larger vessels had bound up in their meshes red corpuscles, but in the capillaries were so closely packed together as to appear almost like a homogeneous mass. In many places the fibrin threads had been deposited thickly outside the vessels, penetrating into the brain matter for a considerable distance. In the vicinity of the blocked vessels were numerous punctate haemorrhages. This deposition of fibrin occurred to a very large extent in all parts of the brain examined, and was far in excess of that which is usually associated with a fibrinous inflammation. The fact has just been alluded to that at times the fibrin is deposited outside the vessels, which implies the passage of fluid contents from the vessels, and their subsequent coagulation. But further, one may also find the other forms of coagula deposited outside the vessels, notably the hyaline spheres, which may undergo still further changes, even becoming vitreous. This was observed in two cases. Calcareous nodules in the grey matter and other parts of the nervous system are not uncommonly found in epileptics. In one case a calcified nodule, the size of a cobnut, was found in the pons Varolii of an epileptic youth just beneath the floor of the fourth ventricle.

To summarise briefly, in the cerebral blood-vessels of epileptics, clots are found which present diverse aspects, but chiefly (*a*) small spherical bodies, singly or in lobulated masses; (*b*) hyaline material clinging to the side of the vessel wall, and more or less occluding the lumen; (*c*) clots of a finely granular material. From the study of the gradational changes, it is believed that the two former arise from the amalgamation of blood plates, while the last results from a disintegration of red blood corpuscles, or from a process of resolution of the hyaline clot.

On evidence obtained from pathological anatomy, taken in conjunction with some experimental procedures, the writer bases

his opinion, that the immediate cause of fits is cortical stasis resulting from obstruction to the blood supply by intra-vascular clots. It is suggested that just before a fit the leucocytes and blood plates shed their nucleo-proteid contents, or that the blood plates simply cohere together. In either case the result is the formation of thrombi of granular or hyaline material, which obstruct the circulation at the place where they arise, or, as free spherical or lobulated masses, they are carried by the blood stream into smaller vessels, in which they become impacted. Probably the larger masses, which form complete casts of the lumen of the vessel, are the result of a more vigorous coagulative process, and this supposition receives support from the fact that they are more commonly met with in cases dying from status epilepticus. Whether a fit ensues after the local obstruction to the blood supply, or not, depends on other factors. The region of the cortex in which the block occurs may have to be taken into consideration. It is not known with certainty whether convulsions will arise from local anaemia of every part of the cortex. Epileptics, it is well known, occasionally appear to miss a period of fits, which are replaced, as it were, by an attack of mania, or a state of stupidity and confusion. These equivalent attacks, it is suggested, may result from plugging of vessels which supply non-explosive (silent) areas of the cortex.

It remains now to ascertain to what extent intra-vascular clotting is met with in other than epileptic brains. For this purpose a series of control brains to the number of thirty from various forms of insanity were examined. General paralytics, in whom epileptiform attacks occur, were excluded, as thrombosis is often found in their cerebral blood-vessels. The result was that while about 30 per cent. of the control cases showed some intra-vascular clotting, in 80 to 90 per cent. of the epileptic cases it was present, and with perhaps one or two exceptions the maximum amount discovered in the non-epileptic was not as much as the minimum found in the epileptic.

The occurrence of some intra-vascular clotting in non-epileptic brains does not affect the validity of the view advocated in this chapter, which assumes that the thrombosis takes place in defectively developed brains, so that there are two factors whose co-operation is necessary before a fit can result:

(*a*) A brain hereditarily and structurally predisposed to instability and convulsion.

(*b*) A sudden deprivation of the normal blood supply.

That blood stasis alone is insufficient appears to be borne out by experiment. Kussmaul and Tenner[1] compressed both carotids in six men, but in two only, symptoms of the nature of an epileptic attack resulted, and *both these cases were of weak intellect.* Hill[2] explains the absence of convulsion after pressure on the carotids by variations in the freedom of the anastomoses in the circle of Willis. From personal observations on the formation of the circle of Willis this explanation seems inadequate. Some variation in the circle is common, but in all, except a few cases, it takes the form of an increase in the calibre of the posterior communicating artery of one side and a slight decrease in the size of the posterior cerebral of the same side, so that in effect the posterior cerebral artery arises directly from the carotid. It is difficult to see how so small a variation could have such a marked effect in modifying the results of pressure. It appears, therefore, more likely that the reason why Kussmaul failed to obtain convulsions in all but the two imbeciles must be sought for in the direction just mentioned, namely, an absence of the first of the required factors.

Summary of the pathological appearances found in epilepsy.

The meninges generally show slight fibroid thickening and a moderate infiltration with leucocytes, chiefly of the large variety. Rupture of the vessels, with extravasation of red corpuscles, and a delicate unorganised foam-like exudate are always present.

Neuroglia. There is an increase of the glia elements, especially those situated in the outer layer of the cortex, and a more or less decided external band of glia fibres, but this is not constant, and is not nearly so marked a feature as some observers contend.

The nerve cells are usually diminished in number—probably in direct proportion to the degree of dementia. The Betz cells are of average number, while they and the larger pyramidal cells are characterised by certain constant features similar to those which are seen in an early stage of the *réaction à distance*, but which represent in these cases an immature form. In all

[1] Kussmaul and Tenner, *New Syd. Soc. Trans.*, London, 1859.

[2] Hill, L., *The Cerebral Circulation*, London, 1896.

cases single nerve cells, or groups of cells, are degenerated and stand out conspicuously from their fellows, in consequence of being shrunken and very darkly stained and with a dense or even homogeneous nucleus. The nucleus varies in different cases and in different cells of the same case; but often, especially in the second, third, and spindle-cell layers, it is swollen, clear, and bladder-like, and is prone to lose its reticular structure, which is replaced by a granular *débris*. The nucleolus is sometimes much swollen. In contradistinction to what one usually finds among imbeciles, the large cells both of the brain and the spinal cord contain a considerable amount of pigment.

The blood-vessels and their contents. The veins are engorged, the capillaries full, and the arteries collapsed and tortuous. In the majority of cases there are no marked structural changes in the walls. The lymph spaces are widely dilated: across them stretches a foam-like exudate, in which are seen large fat drops. Pigment consisting of coarse granules is found in the outer sheath, but chiefly in the vessels of the white matter. As a rule there is very little proliferation of cells in the lymph spaces, and where any such is present it is usually composed of lymphocytes, of which the large varieties preponderate.

Blood plates occur in large numbers, not uniformly distributed but aggregated together.

Hyaline spheres or lobulated bodies lying free in the lumen of vessels are numerous, as also are large hyaline masses forming casts of the vessel in which they lie. Similar appearances are met with in the cerebellar cortex and in the medulla oblongata and spinal cord.

Tract degenerations. Degeneration of the pyramidal tracts, secondary to cortical changes, has been found in slightly less than half, and degeneration of the posterior columns in half the cases examined.

With reference to the *distribution of the lesions* in epilepsy, those occurring in the meninges are widespread, but usually one part shows a greater degree of alteration than another. The cerebellar membranes are often more affected than the cerebral. The glial changes are distributed unevenly over the whole surface of the cerebrum, from the frontal to the occipital poles. Atrophied and sclerosed convolutions are also met with in various regions; the most frequent site is at the anterior end of the cornu Ammonis, but it is a comparatively common

occurrence in the foliae, or even small parts of a single folium of the cerebellum. The next most usual site is probably in the occipital lobe. Although the pathological contents of the vessels are found in all parts, they also are localised, so that whilst certain areas show a large number of affected vessels, adjoining parts may show none.

CHAPTER IX.

THE PATHOLOGY OF EPILEPSY (*Continued*).

Urine—Blood—Cerebro-spinal fluid—Sweat—Thymus gland—Auto-intoxication.

THE FLUIDS AND SECRETIONS OF THE BODY IN EPILEPSY—CHEMICAL PATHOLOGY—AUTO-INTOXICATION.

Owing to the apparent insufficiency of the pathological changes hitherto found in the brains of epileptics to account for the characteristic paroxysmal manifestations, a school of observers broke new ground in their endeavours to find the exciting cause of the severer epileptic convulsions in biochemical changes. In the majority of instances of genuine idiopathic epilepsy, the cause of the recurring convulsions is quite unknown; but numerous observations made during the past twelve or fifteen years seem to suggest that a possible explanation may be at hand in auto-intoxication. The acceptance of this view necessitates a belief in one or other of two important factors: first, the formation of poisonous substances in the gastro-intestinal tract, and their absorption into the blood: or, secondly, the development of one or more toxic agents through disturbance of the bodily metabolism.

The general conclusions, which may be formed from a consideration of the researches upon this subject, are contradictory and confusing, but a *résumé* of the more important work may not be out of place in this chapter.

1. The urine.

The urine of epileptics has been studied from two points of view: (*a*) its constitution; (*b*) its toxicity.

(*a*) *Constitution.* The quantity and colour are normal: the specific gravity is, on the whole, high, even when the total

quantity is large. The reaction is acid. According to some observers (Sala and Rossi [1]) the urea was of average quantity; while others (Alessi and Pierri [2]) found that it was usually under the average amount, and that the variations corresponded with the excretion of phosphoric acid, which, however, varied within normal limits. The chlorides were generally in excess of the normal. Indican was increased after a fit. Albumen, sugar, and peptone were not present in ordinary cases. No connection was found to exist between any variation in these constituents and the occurrence of epileptic seizures.

In contra-distinction to the above, marked changes are found in the excretion of *uric acid*. Both Haig [3] and Krainsky [4] describe a diminution in uric acid excretion during a period of some hours immediately preceding the onset of a fit, to be followed by a well-marked post-paroxysmal increase. Although this obvious alteration in the uric acid quantity exists, there is no evidence in favour of an absolute increase in its production, but only a temporary alteration in its excretion.

Haig lays stress upon the relation of epileptic fits to the normal diurnal variations in the excretion of the uric acid. During the night and early morning hours, when the acidity of the urine is low (alkaline tide), the excretion of uric acid is high; uric acid is in consequence circulating freely in the blood, and the maladies supposed to be associated with circulating uric acid are more common during these hours; viz., migraine, mental depression, and epileptic convulsions. On the other hand, during the "acid tide" of the afternoon and evening, the excretion of uric acid falls, its retention in the tissues takes place, and the rheumatic and gouty pains are thereby developed.

It is unlikely that the retention in the blood of uric acid alone is able to cause epileptic fits, even in predisposed persons: [5] but there would appear to be some foundation for the view that the periodicity of the seizures may be synchronous with the normal variations which take place in the relative alkalinity of the blood. (See p. 107, Chart 11.)

[1] Sala and Rossi, *Gazett. Med.*, August, 1903.

[2] Alessi and Pierri, *Manicomio*, vol. xviii. 1902.

[3] Haig, *Uric Acid*, 1892, p. 21.

[4] Krainsky, *Mémoires Couronnés*, 1901, xv.

[5] According to Bouchard (*Auto-intoxication*, trans., 1894, p. 51), uric acid is not toxic to the extent of ·64 gramme per kilog. of animal injected.

Post-paroxysmal albuminuria. There is much difference of opinion amongst writers as to the presence of an albuminuria as a result of epileptic seizures. Its existence has been denied by some and corroborated by others. The fullest observations are those of Voisin and Péron,[1] who refer to its variable intensity and transitory duration, and whose conclusions are in favour of its dependence upon the degree of renal vasodilatation, as indicated by the cyanosis of the face during a convulsion. On this explanation is founded its constant presence in status epilepticus and in serial epilepsy, its occasional presence with isolated convulsions, and its absence after petit mal attacks.

The observations of Lannois and Mairet[2] are in support of those just given, for they only found temporary albuminuria present in those who became cyanotic during the convulsions.

On the other hand, the statements of Reynolds, Sala and Rossi, and Krainsky, afford no support to these observations.

(*b*) *Toxicity.* As there is great difference of opinion upon the toxic effects of the urine of epileptics when injected into animals, it may be well, in the first place, to ascertain the action of the urine of healthy persons under these circumstances.

The extensive observations of Bouchard[3] may be quoted in this connection. He showed that although the toxicity of normal urine varied with the time of the excretion (whether day or night, or after exercise), its injection into animals was followed by toxic symptoms, and that the toxic dose corresponded to about 45 c.c. per kilogram of the animal injected. He showed that amongst the toxic principles of the urine were a narcotic substance, and two convulsion-producing bodies, potash, and an organic substance, probably of alkaloidal nature.

Roncoroni[4] also stated that normal human urine, when injected subcutaneously into rabbits, produced symptoms of mild toxaemia such as lethargy, prostration, and a feeble response to external stimuli. Sometimes it led to the death of the animal.

It is therefore evident that the toxic effects produced by

[1] Voisin and Péron, *Arch. de Neurologie*, vol. xxiii. p. 353.

[2] Lannois and Mairet, *Lyon. Médicale*, 1899, p. 365.

[3] Bouchard, *Auto-intoxication*, trans., 1894, p. 51.

[4] Roncoroni, *Archivio di Psichiat*, 1900, xxi.

the injection of urine into an animal may be induced even by the excretion of a healthy person.

The observations upon the toxicity of the urine of epileptics have extended over a number of years, since Bouchard (1885) and Denne and Chouppe (1889) originally recorded their experiences. But it is mainly through the work of Voisin and Péron[1] that this subject was elaborated. These observers showed that immediately prior to a fit there was urinary hypotoxicity, gauged by injections into rabbits, that during the seizures the toxicity rose, and reached a point considerably above normal after the fit—hypertoxicity. During the interparoxysmal periods the toxicity was comparable to that of normal persons. If after a seizure the toxicity did not rise, a continuance of the seizures, or even maniacal excitement, might be expected. Certain epileptic dements showed a constant hypotoxicity, a feature which was also present during some of the mental states of epilepsy. Upon these observations the authors explained certain clinical phenomena, such as the pre-paroxysmal furred tongue and loss of appetite, and the other premonitory symptoms of an epileptic seizure. Following the seizure ensued polyuria, sweating, and sometimes diarrhoea, indicating the discharge from the organism of the toxic matter. If this elimination did not occur, status epilepticus, and sometimes death, ensued. Assuming these observations to be correct, a plausible and seemingly satisfactory explanation of serial epilepsy and the status epilepticus was at hand.

The researches of Mairet and Vires[2] upon urinary toxicity showed that so constant was the interparoxysmal hypotoxicity in epileptics as almost to constitute a permanent and characteristic feature of the disease.

On the other hand, there is a considerable mass of evidence against the constant presence of these conditions. Working with subcutaneous injections into guinea-pigs of the fresh, filtered urine of epileptics, Roncoroni[3] obtained results in many cases comparable to what was found after injection of normal urine: sometimes death resulted, at other times only symptoms of a mild toxaemia. He formed the general conclusion that the phenomena were not so much the consequence of the

[1] Voisin and Péron, *Arch. de Neurolog.*, vol. xxiv. p. 178.

[2] Mairet and Vires, *Comptes Rendus*, 1898, June 25.

[3] Roncoroni, *Archivio di Psichiat.*, 1900, xxi.

urinary toxicity, as dependent upon the disposition (idiosyncrasy) of the individual animals, subject to the injections. The toxicity was only in some cases really increased: in most the urine of epileptics was not more toxic than that of health. When the toxicity was clearly increased, it was an open question whether it could be regarded as a cause of the fits.

Hebold and Bratz[1] also experimented along similar lines with dogs and white mice. The urine of the two periods, immediately, and some hours, after a fit, and from cases of status epilepticus, when subcutaneously injected, did not give rise to phenomena, differing from those produced by the injection of normal urine. If positive results were obtained there was no constancy in their repetition.

2. The blood.

The blood in epilepsy may be considered from the same points of view as the urine: (*a*) its constitution, (*b*) its reaction, (*c*) its toxicity.

(*a*) *Constitution.* The general characters of the blood of epileptics, as given by Pearce and Boston[2] are those of a fairly well-marked chlorotic type of anaemia: haemoglobin 62·8 per cent., red corpuscles 4,500,000, and specific gravity 1·05. The red cells sometimes show a moderate poikilocytosis. The polymorphonuclears may be reduced in number. A slight leucocytosis is an almost constant feature.

After the injection of small quantities of blood from epileptics into rabbits, these observers found that the haemoglobin rose from 65 to 84 per cent., that the leucocytes increased from 8000 to 17,000 per c.mm., and that the red cells fell considerably in number. Other observers have also found considerable leucocytosis after epileptic convulsions, more especially of the eosinophile cells. The specific gravity of the blood is also stated to rise from 1·056 to 1·059 after fits.

Leucocytes. The relation of the leucocytes to epilepsy has been the subject of considerable investigation, more particularly within the last few years. M'Phail[3] stated that these cells were numerically about 20 per cent. below the normal, but that under the influence of bromide of potassium they approached

[1] Hebold and Bratz, *Deut. Med. Wochensch.*, 1901, m. xxxvi.

[2] Pearce and Boston, *American Jour. of Insanity*, 1904.

[3] M'Phail, *Jour. of Mental Science*, 1884, vol. xxx.

more nearly to the normal. Pugh's[1] observations were made more particularly with relation to the fits, and indicated that after a fit a marked leucocytosis occurred, both of the large and small lymphocytes, as well as of the eosinophiles; and that in the interparoxysmal periods and in status epilepticus there were wide variations in their number. The recent observations of Onuf and Lograsso[2] on this subject show that there are daily variations in the number of leucocytes in epilepsy, not entirely explainable by the influence of meals, rest, or work. There may be a leucocytosis before an attack, but not necessarily, nor is there any direct relation between the fit and leucocytosis, which latter may indeed occur independently of a seizure. Lewis Bruce[3] found that every case of epilepsy showed hyperleucocytosis during the periods when the patients were suffering from seizures, but also in the intervals when they were quite free. The most marked period of hyperleucocytosis followed a fit, or occurred during the period when the patient suffered from a series of seizures. In cases of epilepsy with maniacal excitement, occurring either as postparoxysmal or equivalent symptoms, he[4] found marked hyperleucocytosis amounting to 30,000 per c.mm., along with quickened pulse and some elevation of temperature, features which he considers diagnostic of toxaemia. Dr. John Turner states that blood film preparations usually showed a considerable leucocytosis and increase of the blood plates, but that there was no apparent difference in their numbers whether the films were taken during a fit, or in the interparoxysmal period.

Blood plates and *blood coagulation.* There is no satisfactory method of estimating the number of blood plates, but it has been stated by Determan that they average about 1 to every 22 red cells, or about 300,000 per c.mm. One of their features is that they adhere rapidly to each other when blood is withdrawn from the body. This rapid cohesion and amalgamation has been observed within the cerebral blood-vessels, and described in the chapter upon Pathology, where it is contended that they play an important part in the formation of the intra-vascular thrombi. There is some evidence in support of the view

[1] Pugh, *Brain*, 1902, vol. xxv.

[2] Onuf and Lograsso, *Amer. Jour. Med. Sciences*, Feb. 1906.

[3] L. Bruce, quoted by Macpherson, *Jour. of Ment. Science*, Apr. 1905.

[4] L. Bruce, *Studies in Clinical Psychiatry*, 1906, p. 169.

that an increased tendency to blood coagulation exists in epilepsy. From observations, which have been made for me by Dr. John Turner, a considerable difference in the clotting time before and after a fit is shown. The blood from 26 epilepties was examined on 118 occasions, and the mean coagulation time was found to be 120 seconds. The mean coagulation time in 13 cases of insanity on 38 occasions was 136 seconds. He found that the coagulation time in epileptics was materially diminished just before, during, and shortly after the convulsive seizures. This was noted to have been the case in 19 instances out of 23, who had fits during the period of examination. There was a difference of from 30 to 90 seconds in the coagulation time, soon after the fits and in the interparoxysmal periods.

Clinical evidence also lends support to the view that there is an increased tendency in epilepsy to blood coagulation. It is well known that epileptics recover rapidly from severe wounds, even under unfavourable conditions.

In this connection the results of some experiments conducted by Tirelli[1] upon the blood serum of epileptics may be quoted. He found that the serum was influential in attenuating and delaying the virulence of the staphylococcus pyogenes aureus, and to this property he ascribed the invulnerability of epilepties to severe injuries.

(*b*) *Reaction.* The blood is normally slightly alkaline in reaction, but it undergoes variations in the degree of alkalinity from various physiological and pathological processes.

The observations of Charon and Briche,[2] upon the reaction of the blood in epileptics, showed the relation of epileptic convulsions to the normal variations, whereby the seizures were found to stand in inverse relationship to the degree of blood alkalinity, the minimum frequency of the attacks corresponding to the maximum blood alkalinity, and *vice versa.* The researches showed that the blood alkalinity attained its minimum in the late evening and remained stationary during the greater part of the night, during which time epileptic seizures are fairly frequent (chart 11).

In consequence of these observations they attempted to control the fits by maintaining the standard of alkalinity through

[1] Tirelli, *La Riforma Medica*, 1902, vol. iv. n. 50.

[2] Charon and Briche, *Arch. de Neurolog.*, 1897, No. xxiv.

repeated injections of alkaline solutions. The general results of these endeavours, however, lay in diminishing the number of isolated seizures and of increasing the tendency to serial attacks.

Of greater importance in this connection are the researches of Pugh[1] upon the reaction of the blood in immediate association with the seizures. This observer found that the average interparoxysmal alkalinity was lower than the average of the control cases; that a sudden and well-marked fall in alkalinity occurred immediately prior to the onset of a fit, and that a further diminution took place after the seizure. The explanation, which he offered for the diminished alkalinity, lay in the accumulation of acid toxines due to absorption from the intestinal tract of lactic, acetic, and butyric acids. On the other hand, he regarded the pre-convulsive fall in alkalinity as a "biochemical aura"; while the post-paroxysmal further diminution, not however found after petit mal attacks, was directly due to the accumulation of the acid products of muscular metabolism (carbonic and sarcolactic acids). This varied considerably with the number and intensity of the seizures.

(*c*) *Toxicity.* The injection of the blood of a healthy animal into the veins of another animal may produce death, without it being attributable to embolism. The injection of 25 c.c. of blood per kilogram of animal invariably causes death (Bouchard). The toxic influence of injected blood varies according to the part of the vascular apparatus from which it is withdrawn, and according to whether it is injected into similar, or dissimilar, species of animals. The blood toxicity of man varies from 15 c.c. per kilogram (Mairet and Vires) to 30 c.c. (Herter).

The observations upon the toxicity of the blood of epileptics vary considerably; some writers holding that it is less toxic (Mairet and Vires),[2] others that there is no difference (Herter,[3] Hebold, and Bratz), while a third series describe an increased toxicity (Cololian,[4] Krainsky[5]).

The experiments of Krainsky[5] are the most complete as yet recorded upon the auto-intoxication theory of the production

[1] Pugh, *Brain*, 1902, p. 500.

[2] Mairet and Vires, *op. cit.*

[3] Herter, *Jour. Neuro. and Ment. Diseases*, 1899, p 72.

[4] Cololian, *Arch. de Neurolog.*, vol. vii. p. 177.

[5] Krainsky, *Neurol. Centralbl.*, 1897, p. 697.

of epileptic seizures. He is in general agreement with Haig upon the marked fall in the uric acid exeretion beforc a fit, and its rise afterwards. He found also a constant relation between the daily quantity of exereted urie acid and the number and intensity of the seizures.

He found that subeutaneous injeetions of 1-3 e.mm. of blood taken shortly before a single attack, and from cases of status epileptieus, were followed by paralysis and epileptie eonvulsions and death of the animal in four or five days. Injeetions of blood from the interparoxysmal period gave no sueh results. He eoneluded from his experiments that the blood of epilcptics contained a poison, whieh gave rise to rccurring eonvulsions. He claimed that the poison was the carbamate of ammonium, whose acid radiele is earbamie aeid, whieh breaks up into urea by the abstraetion of a molecule of water. He stated that injeetions of ammonium earbamate into animals produced symptoms eomparable to those induced by the injeetions of the blood of epileptics. In a further series of experiments, Krainsky found earbamie acid present in the blood of epileptics in eonsiderable quantities. Upon this foundation he treated epileptics with lithium earbonate, and found it temporarily efficaeious.

The presence of a parasite in the blood of epilepties has been described by Bra,[1] who has applied to it the term *Neurococcus*. Further investigation upon this point has not been eonfirmatory, both Besta[2] and Tirelli and Brossa[3] regarding its existence, as described by Bra, as due to defeetive teehnic. On general grounds it is scareely eoneeivable that epilepsy and its phenomena can be referred to the presenee of a special micro-organism.

3. Cerebro-spinal fluid.

Cholin, a substanee found by Mott and Halliburton[4] in thc cerebro-spinal fluid in cases of nervous disease eharaeterised by myeline disintegration, has also been found in the fluid of epileptics by Donath,[5] S. A. K. Wilson, and others. Donath found it in 19 out of 22 eases of epilepsy examined for the purpose.

[1] Bra, *Rev. Neurol.*, x. 1902.

[2] Besta, *Riv. Sper. di Freniatria*, 1902, p. 309.

[3] Tirelli and Brossa, *La Riform. Med.*, 1903, No. xxxiv.

[4] Mott and Halliburton, *Brit. Med. Journal*, 1904, ii. 1557.

[5] Donath, *Deutsch. Zeitsch. f. Nervenh.*, xxvii. pt. 1.

Some observers hold that it is the cause of epileptic convulsions, its presence acting as a toxic agent, as it has also been found in cases of general paralysis with epileptiform seizures. On the other hand, it has been regarded as the result of the seizures, and in no way should it be considered as a causal factor. Moreover, there is evidence that a trace of cholin is a normal constituent of cerebro-spinal fluid; for Mansfeld[1] has shown that, both in normal human urine and in the cerebro-spinal fluid of man, crystals identical with Donath's cholin chloro-platinate have been observed.

Experimental evidence upon the injection of cholin into animals is also of a contradictory nature. Dide[2] obtained the following results from the injection of the cerebro-spinal fluid of epileptics into the brains of rabbits:

(*a*) If taken between the seizures and injected no result ensued.

(*b*) After an isolated fit, injections produced slight lethargy, stupor, or sometimes general tremors—the symptoms being only of temporary duration.

(*c*) After a series of attacks, small doses gave rise to general twitchings, and larger doses resulted in the death of the animal in an epileptic fit within a few minutes.

On the other hand, there are the observations of F. Buzzard and Allen,[3] who found that repeated injections of moderate doses of cholin into the circulation produced neither convulsions nor paralysis; that large doses produced convulsions, but that such doses were relatively much in excess of what could be produced in man under ordinary degenerative conditions; and that it is improbable that the convulsions of general paralysis and of epilepsy are directly, or solely, due to the presence of cholin in the blood or cerebro-spinal fluid.

That cholin is absent from, or only present in minute traces in, the cerebro-spinal fluid in cases of functional nervous disease, such as hysteria, has also been proven; a fact which has been used to differentiate the organic from the functional disorders.

S. A. K. Wilson[4] has shown that cholin stands in no relation to lymphocytosis of the cerebro-spinal fluid, for, in cases with

[1] *Neurol. Centralbl.*, 1905, p. 221.

[2] Dide, *Neurol. Centralbl.*, 1902, p. 129.

[3] F. Buzzard and Allen, *Rev. of Neurology*, vol. iii. p. 453.

[4] *Neurol. Centralbl.*, 1905, p. 687.

pronounced lymphocytosis, cholin has been absent; and, on the contrary, cholin has been present without any lymphocytosis.

The observations of Merzbacher[1] have shown that lymphocytosis is not a constant feature in the cerebro-spinal fluid of epileptics; for out of twelve cases which he examined for the purpose, it was only present in six, four of which were cases of traumatic epilepsy; four presented no sign of lymphocytosis, and in the remaining two it was present to only a slight extent.

The explanation of the presence of cholin in epilepsy is a debatable point. There is no known disintegration of the medullated fibres in the early stages of the disease; but the investigations of Dr. John Turner have pointed to the existence of thrombotic occlusions as possible causes of some epileptic seizures, and more especially of the acuter forms, such as serial epilepsy and the status epilepticus. Among the results brought about by these lesions are breakings up of the medullated fibres of the cortical association tracts, and the fibres of the cornu Ammonis; disintegration of which in course of time leads to sclerosis of several portions of the cortex, and occasionally to degenerative changes in the spinal cord (p. 181).

4. The sweat.

The sweat of epileptics is considered to possess a toxic property like the urine and the blood. Cabitto[2] has stated that sweat from epileptics during the period prodromal to a seizure, if injected into rabbits, has a toxic action, and gives rise to convulsion; that this toxic property increases as the attack advances, and decreases after the paroxysm; and that during the interparoxysmal period no such toxic influence is observed. On the other hand, Mavrojannis[3] found that the action of the post-paroxysmal sweat upon rabbits was entirely negative.

5. The thymus gland.

Ohlmacher[4] described the presence of an enlarged and persistent thymus gland in a number of cases of epilepsy, along

[1] *Neurol. Centralbl.*, 1904, p. 555.

[2] Cabitto, *Riv. Sperimental di Freniatria*, 1897, p. 36.

[3] Mavrojannis, *Rev. de Psychiatrie*, 1898, p. 199.

[4] Ohlmacher, *Bulletin, Ohio Hospital*, 1898.

with a hyperplastic condition of the lymphatic structures of the body. The thymus gland usually attains its full development at birth, and remains stationary until about puberty, when atrophy commences. It is stated as of some significance that the onset of epileptic seizures is most common during the period of the thymus gland's greatest development and activity; but that after twenty years of age, when the gland has disappeared as a functionally active structure, the onset of epilepsy is rare. This gland would appear to be mainly concerned in the metabolism of nitrogen compounds, as it is known to contain large quantities of purin bodies (Walker Hall),[1] these substances being increased in the urine upon a diet of thymus gland. Observations have shown that preparations of thymus gland given to epileptics materially aggravate the disease.[2]

Is there a type of epilepsy due to auto-intoxication ? The discrepancies which exist in the observations upon urinary and blood toxicity in epilepsy do not afford a thoroughly satisfactory basis upon which to build a theory of auto-intoxication. Nor has it yet been clearly demonstrated whether the toxic condition of these fluids, assuming them to be toxic to some extent, stands as cause, or effect of the convulsions. Some explanation of the variations recorded by observers may be ascribed to differences in the nature of the cases upon which the researches were carried out. It is not indeed contended that attacks of petit mal, or the minor type of epilepsy, are associated with any of the toxic qualities of blood and urine just described. Nor is it apparent from the observations that the average type of epileptic fit, characterised by frequently recurring seizures of a medium convulsive charaeter, is to be ascribed to toxaemic influences. Those cases which have given the most pronounced reactions, both of the blood and the urine, were examples of serial epilepsy, the status epilepticus, and fits associated with aeute mental symptoms; types of the disease in which there is more reason to suspect a probable auto-intoxication, on account of the general somatic disturbances which accompany them.

As already pointed out, when the clinical types of epilepsy were detailed, and as the observations upon the urinary

[1] Walker Hall, *Purin Bodies*, London, 1903.

[2] Hodgson, *Medical Chronicle*, 1903, p. 14.

and blood toxicity have revealed, certain general features necessarily co-exist, if infection, or intoxication is the causal agent. Briefly these are :

(*a*) Well-marked premonitory signs.

(*b*) Fits recurring frequently and developing either into serial epilepsy, or the status epilepticus.

(*c*) More or less prolonged intervals of freedom between the attacks.

One would *à priori* suppose that attacks likely to arise from intoxication would assume the characters above described, and Binswanger has already pointed out that a small group of epilepsies, characteristic of intoxication, may be detected in prolonged series of attacks, followed by long periods of freedom.

A generally recognised feature of infection is found in increased coagulability of the blood, a fact which has been shown to occur readily in some forms of epilepsy. Intra-vascular clotting occurs clinically in cases of infection, most probably as a result of the liberation of nucleo-proteid ; and Wooldridge showed that the intra-vascular injection of nucleo-proteid caused clotting in the blood-vessels during life, without any accompanying injury or disturbance of the vessel walls. Now, it has been already shown, when discussing the pathological findings in the brains of persons who have died of epileptic seizures, that nucleo-proteid clotting is not uncommon, from which circumstance it may be inferred that some types of epileptic convulsion are due to nucleo-proteid thrombosis, arising from toxaemic influences within the body, or ascribed to absorption from the alimentary canal.

A further sign of toxaemia, described by Lewis Bruce as occurring in some forms of insanity of toxic causation, is a hyperleucocytosis associated with a high percentage of polymorphonuclear cells. Assuming that the normal leucocyte count is about 8000 per c.mm., an increase up to 15,000, or even 30,000, is not infrequently seen in the acute toxaemic insanities. In epilepsy the leucocytosis has been variously stated, but it would appear from the observations of Pugh and Bruce that a hyperleucocytosis was especially to be found in serial epilepsy and cases complicated with acute mental disorder.

When these observations are considered in conjunction with

the increased tendency to intravascular coagulation and thrombosis, which have been described in Chapter VIII., there would appear to be proof that some types of epileptic paroxysm may be ascribed to auto-intoxication, in persons hereditarily and structurally predisposed to convulsion. These are, serial epilepsy, the status epilepticus and fits associated with acute psychoses as postconvulsive symptoms.

CHAPTER X.

THE DIAGNOSIS OF EPILEPSY AND OF EPILEPTIC FITS.

Diagnostic features—Epileptic and hysterical fits—Epileptiform convulsions—Aural vertigo—Fits in adult life.

THE DIAGNOSIS OF EPILEPSY.

The diagnosis of the epileptic "symptom-complex" is based upon a number of factors. In the main these are the epileptic character and temperament, the symptoms and signs of an inherited degenerative disposition, the type of the seizures with their immediate sequelæ, and the interparoxysmal mental condition.

As both the epileptic disposition and the inter-paroxysmal mental condition have been already fully described, it is only neeessary in the present chapter to refer to those features, which are eharacteristic of epileptic seizures, and to indicate the points by which they may be distinguished from some other paroxysmal phenomena.

What are the features by which an epileptie seizure may be reeognised ?

1. *Sudden loss or obscuration of consciousness.* The existence of this phenomenon at some period of the disease is essential to the diagnosis of epilepsy ; although, as we have already shown, when detailing the clinical characteristics of fits, each and every epileptic manifestation is not necessarily associated with a loss of conseiousness. Some incomplete seizures (aura fits) are unaeeompanied by it, but when these pass into the more complete attaek, eonsciousness is obscured or lost. Loss of eonsciousness is reeognised most readily by the abolition of the eorneal and of the pupillary light reflexes, symptoms which lend themselves readily to examination. The reappearance of these reflexes is synehronous with the return to consciousness after the fit.

Sometimes the loss of consciousness is only momentary, leading to a fall without convulsion and followed by rapid recovery.

2. *A sudden fall*, from obscuration of consciousness, is an epileptic phenomenon; but some epileptic paroxysms are unaccompanied by a fall, such as many forms of minor epilepsy and most psychical attacks.

3. Highly characteristic of epilepsy, but only observed in the more fully developed seizures is *the sequence of tonic spasm and clonic convulsion.* Hence wetting the clothes, or the bed, and tongue-biting, indicate that with the loss of consciousness, there has also been spasm and convulsion. The bladder is usually emptied at the commencement of the tonic stage, and the tongue is bitten during the period of clonic convulsion. Clonic convulsion, without loss of consciousness, or antecedent tonic spasm, is not usually found in idiopathic epilepsy, but is more characteristic of epileptiform seizures (Jacksonian epilepsy) arising from local organic cortical disease.

4. Some miscellaneous phenomena, observed after fits, suggest their epileptic nature. Of these subconjunctival ecchimoses, dislocation of the shoulder, subcutaneous effusions of blood, and scars or other evidence of injury, recent or remote, acquired during "an attack," may be mentioned.

5. *Fits coming on during sleep* are almost always epileptic. As fits during sleep may pass unrecognised for many years, it is important to ascertain whether there are any symptoms by which their existence may be established. In addition to those already mentioned, are the following; repeated falling out of bed, periodic headache accompanied by a feeling of lassitude, heaviness, and a sensation of having been bruised or beaten all over, on waking in the morning; and occasional soreness of the mouth or tongue, with perhaps slight blood staining of the pillow.

Voison mentions the following phenomena as highly suggestive of epilepsy, more especially the nocturnal form: intermittent wetting of the bed, occasional periodical and temporary mental dulness and irritability, well-marked lapses of memory, somnambulism and unexplainable outbursts of temper.

A form of periodic seizure, occurring during sleep, found more particularly in young people or in early adult life, resistive to treatment by the bromides, and often unassociated with attacks of any sort during the day, is the loud piercing scream, which may persist for many years, and which appears to be of epileptic nature

Notwithstanding these readily recognised features of epilepsy, instances occur in which it is scarcely possible to state definitely whether recurring attacks are of an epileptic nature, or are symptoms of some other malady—hysteria, aural vertigo or epilepsy with localised convulsion—in which periodic "attacks" are a prominent symptom. In such cases the diagnosis remains for a time uncertain, until the further course of the malady ultimately reveals the true nature of the seizures. In idiopathic epilepsy there develop in time the mental phenomena of the interparoxysmal periods, and the paroxysmal psychoses, either as post-convulsive or as equivalent states. These are characteristically absent in hysteria.

1. Epileptic and hysterical seizures.

An important matter is the differential diagnosis between epileptic and hysterical attacks. As both epilepsy and hysteria are diseases based upon a hereditary degenerative foundation, there are certain clinical symptoms common to both, viz., the periodic recurrence of paroxysmal attacks, which present many analogous features. There may also exist in the same person genuine epileptic phenomena with hysterical manifestations; and there has been stated to occur a mixed form—the so-called *hystero-epilepsy*.

The most recent writings on this subject, however, tend to show that there is no such generally recognised condition as hystero-epilepsy in the usually accepted sense. Bratz and Falkenberg[1] from a study of 2500 cases of epilepsy, found no instance of hystero-epilepsy, *i.e.*, a mixed form, in which from the commencement of the disease there are epileptic and hysterical symptoms giving character to both the paroxysmal and inter-paroxysmal states. All their cases were either simple epilepsy or hysteria, or a combination of both; the co-existence of the two being not at all rare (38 cases out of 734, or 5 per cent).

Other observers conclude that there is no single mark of differentiation between the epileptic and the hysterical seizure, neither the state of consciousness, nor the alteration of the reflexes. Both disorders are characterised by major and minor seizures, occurring singly, or in series, and in the inter-paroxysmal stage of both, prolonged states of ambulatory automatism may be found.

[1] Bratz and Falkenberg, *Arch. f. Phych.*, 1903, p. 328.

The question therefore arises, Is it possible to draw any distinguishing line between epilepsy and hysteria as morbid conditions, and between the paroxysmal phenomena of the two disorders? The answer to the first question lies in a study of the course of the two diseases, epilepsy being a progressive degenerative malady ending usually in dementia; while to the second, an answer will forthwith be given which will aid the observer in differentiating the purely epileptic from the purely hysterical fits.

The complete epileptic fit being a "discharge" from cortical areas, pursues a well recognised march, and is followed by a train of immediate sequelæ, resulting from inhibition, exhaustion, or temporary functional depression of the cortical neurones.

In the hysterical attack, on the other hand, there is no such march and no well defined sequelæ referable to changes in the motor paths.

Upon this basis a differential diagnosis may in many cases be made, and the distinguishing phenomena studied according as they are observed during, or after, the fit.

1. *Paroxysmal phenomena—the fit.*

Epileptic.	Hysterical.
Onset—sudden, sometimes with a cry and fall; frequently with an aura.	Gradual—fall rare, and cry a long sustained wail.
Consciousness—abolished, shown by loss of corneal reflex.	Retained or perverted: corneal reflex present.
Convulsion—tonic stage of short duration, followed by clonic stage involving limbs on one side more than the other. Biting of the tongue.	Tonic spasm with opisthotonos, or struggling and wild purposive movements requiring restraint, or quick and rapid clonic tremor of limbs. May bite the onlookers.
Pupils—dilated and immobile during convulsion.	Pupils react to light in minor attacks: probably inactive in hysteria major.
Termination of fit through stage of exhaustion and stertor.	Sudden as a rule; fit can be cut short by faradisation.
Evacuation of bladder and sometimes of rectum during tonic stage.	Never involuntary evacuation of bladder.
Duration: 30 seconds to 2 or 3 minutes.	A few minutes to 20 or 30 minutes.

The above points form the salient elements in the differential diagnosis. If an attaek is being described by an untrained onlooker, it may be a difficult matter for the physician to decide whether the paroxysms are those of epilepsy or hysteria, as the duration of the minor seizures in both diseases is nearly the same, and they may both reeur in definite series. Moreover, the perversion, or dissoeiation, of conseiousness in hysteria, may readily be mistaken for its abolition, which is so characteristie of the epileptie attaek.

On the other hand, should the attaek be observed by the physieian, there is as a rule less difficulty in differentiating the two. The rolling or squinting movements of the eyeballs, and the oseillations of the head in hysteria, are quite distinet from the tonie eonjugate deviation of the head and eyes in epilepsy; the tonic spasm of the limbs inducing flexion of the arms and extension of the legs in the latter, are in eontrast to the tonie spasm mainly of the muscles of the neck and baek in the former; the steady development and eventual yielding of the elonie spasms in epilepsy stand out in striking eontrast to the vibratory tremors of the hysterieal attack; while the sudden relaxation of spasm and the almost instantaneous return to eonseiousness in the hysterieal seizure is a feature of noteworthy importanee.

Should the pupillary light reflex be in abeyanee in an attaek presenting hysterieal features, we have probably to do with one of the so-ealled hystero-epileptie seizures deseribed by Chareot, Féré, and others of the Freneh sehool, but whieh are undonbtedly rare in this eountry.

2. *Post-paroxysmal sequelæ.*

Epileptic.	Hysterical.
Motor paralysis, uni- or bilateral, of a temporary character.	No motor paralysis.
Sensory paralysis, giving a uni- or bilateral hypæsthesia and hypalgesia.	No sensory paralysis, except what may be found as an inter-paroxysmal permanent phenomenon.
Tendon-jerks abolished or increased; the latter sometimes on one side only.	Tendon-jerks normal or increased bilaterally.
Ankle clonus on one or both sides according as the fit was mainly uni- or bilateral.	No ankle clonus, except sometimes of a spurious kind.

Epileptic.	Hysterical.
Plantar reflex, flexion or extension ; sometimes both.	Plantar reflex, never extension ; may sometimes be absent.
Abdominal reflexes, absent on one or both sides.	Abdominal reflexes retained.
Temporary automatism.	No post-paroxysmal automatism.

The alteration in the reflexes, as above mentioned, may form an important element in the differential diagnosis between epileptic and hysterical attacks. If the convulsions in epilepsy have been more pronounced on one side, as is commonly the case, the reflex phenomena may present temporarily, features similar to what are observed with a permanent organic lesion of the pyramidal system—increased knee-jerks, ankle-clonus, plantar extension and abolition of the abdominal reflexes.

Although the above mentioned phenomena may be regarded as the lines along which the differential diagnosis may be made, many writers are disinclined to state whether there is any single mark, by which the two forms of seizure may be distinguished. Bratz, Falkenberg, and Binswanger, believe that neither the state of consciousness, the condition of the pupil, the knee-jerks, the tongue-biting, or the occurrence of fits during sleep, are really sufficient to establish the diagnosis of epilepsy.

A method of differentiation has accordingly been invoked in *hypnosis*, whereby the state of consciousness during the paroxysmal period in hysteria may be recalled. The occasional apparent success of hypnotic suggestion in the treatment of epilepsy may possibly be ascribed to the assumption that the cases were of hysterical rather than epileptic nature.

2. Epilepsy and localised convulsions (Jacksonian Epilepsy).

The leading points of differential diagnosis, which distinguish the localised or epileptiform convulsion, from those of a general epileptic character are, the absence of a tonic stage, and the onset of unconsciousness (if it occurs at all) at a late period of the fit. In Jacksonian epilepsy the convulsion starts in clonic spasms, which are sometimes preceded by an aura in the affected limb, invades the remainder of the limb and perhaps the whole of one side of the body, before it passes to the limbs of the opposite side and becomes generalised, when loss of consciousness ensues.

The commencement of an attack of idiopathic epilepsy, with

a peripheral warning and tonic spasm of a limb preceding the loss of consciousness—Jacksonian onset of an epileptic fit—may present difficulty in the diagnosis for a time, as it is a not uncommon type of epileptic seizure; but I cannot confirm Collier's statement that the commonest cause of Jacksonian convulsion is idiopathic epilepsy. The separation of these two types of seizure is in some cases only possible by a study of the associated phenomena, and the further progress of the malady. Along with the localised convulsions of organic cerebral disease there will be found co-existent, or sequential, paralysis of a mono- or hemiplegic type, and general symptoms of gross intracranial lesion, such as headache, vomiting and optic neuritis; while in the idiopathic malady there will later on appear the characteristic mental phenomena of the inter-paroxysmal stages.

The investigation of the personal and family history of the patient is also of much value in diagnosis. It is rare to find a family history of epilepsy in genuine cases of Jacksonian epilepsy. As the latter is caused most commonly by syphilitic lesions of the membranes of the brain, a history of previous infection is an important point; while the therapeutic test is one which may throw light on a case, which is in other respects of doubtful nature.

Reference has been made on a previous page to the convulsions of a general epileptic type occurring in consequence of organic cerebral disease.[1] These are the cases of so-called "organic epilepsy," in which the fits do not differ in any essential feature from those of idiopathic epilepsy, except perhaps in a constant local commencement and in a greater implication of one side of the body, and the persistence, after the seizure, of a more permanent motor paralysis. Collier[2] has drawn attention to the large number of patients, suffering from symptoms apparently conclusive of idiopathic epilepsy, but who later on develop other signs of intracranial new-growth.

3. Minor epilepsy and aural vertigo.

There is usually little difficulty in distinguishing between an epileptic seizure and the severe attacks of labyrinthine vertigo. The suddenness of the onset, the intense giddiness hurling the

[1] *Vide ante*, p. 60, and later on under Surgical Treatment.

[2] Collier, *Brain*, 1904, p. 498.

victim forcibly on to the ground, the severe and sometimes prolonged vomiting, the retention of consciousness, the slow and gradual recovery, extending over several hours or days, form a clinical picture of a characteristic type. Associated also with such attacks are the physical signs of nerve deafness, in one or both ears, and tinnitus aurium.

It is, however, in the slighter forms of aural vertigo that a difficulty may arise, as these cases sometimes exhibit periodic attacks of loss of consciousness, often with a fall, indistinguishable from some forms of minor epilepsy.

Gowers[1] has directed attention to the features of the brief attacks of loss of consciousness, which may be a puzzling symptom of aural vertigo. As he points out, vertigo is in itself an interference with normal consciousness, so that any sudden increase in its intensity may lead to loss of consciousness and a fall. Even with the associated symptoms of labyrinthine disease, which these cases present, it may be difficult to state that the attacks are not of an epileptic nature.

The resemblance, indeed, between these attacks and minor epilepsy is marked, even to the temporary pallor of the face. It would therefore be more accurate to describe them as epileptoid phenomena. In the cases of this nature, which I have seen, there was no family history of epilepsy, there were no physical or mental stigmata of a degenerative character, and there was an absence of those phases of automatism, which so commonly accompany, or follow, the lapses of consciousness in the idiopathic disease. The points of resemblance and of difference are stated in the subjoined parallel columns.

Minor Epilepsy.	Minor Aural Vertigo.
Onset sudden.	Onset sudden.
Duration brief.	Duration brief.
Temporary loss of consciousness: pallor of face.	Temporary confusion or loss of consciousness: perhaps pallor of face.
Fall, more or less sudden.	Fall, with or without feeling of being forced or drawn down (Gowers).
Frequently post-paroxysmal automatism.	Never automatism.
Sometimes auditory aura: no deafness.	Tinnitus and signs of labyrinthine deafness.
Interparoxysmal mental state, as already described.	No impairment of memory, or dementia.

[1] Gowers, *Brit. Med. Journ.*, 1906, ii. p. 7.

In conclusion, brief reference may be made to the diagnostic value of **a convulsive attack occurring in adult life,** for instance, between the ages of thirty five and fifty. Such a seizure may be the onset of late epilepsy, but before this diagnosis is accepted, an examination into the following conditions should be made, and their possible existence eliminated:

1. A syphilitic affection of the cerebral meninges.
2. The onset of general paralysis of the insane.
3. The presence of an intracranial tumour.
4. Chronic renal cirrhosis.

CHAPTER XI.

THE PROGNOSIS AND CURABILITY OF EPILEPSY.

Statistics of cure—Prognosis based upon treatment—Conditions influencing prognosis—Heredity—Age at onset—Duration of the disease—Frequency of seizures—Character of seizures—Sex—Cure of epilepsy—General conclusions.

I. PROGNOSIS OF EPILEPSY.[1]

Much attention has been given from classical times onwards to the prognosis of epilepsy. It would, however, serve no useful purpose to refer to the numerous authorities on this subject, or even to briefly state their conclusions. An exception ought, however, to be made in the case of Hippocrates, in whose writings will be found much that is of value even at the present time. For example, when describing the "Sacred Disease" he wrote:[2] "If it attacks little children the greater number die. . . . If youths and young adults, recovery may take place, but there is danger of its becoming habitual, and even increasing if not treated by suitable remedies. Such also is the case when it attacks children. . . . When it attacks people of advanced years it often proves fatal. . . . When a person has passed the twentieth year of his life the disease is not apt to seize him, unless it has become habitual from childhood. . . . When the disease has prevailed for a length of time it is no longer curable."

It would appear, however, as if the ancient Greek and Roman physicians took a more generally favourable view of the disease than has been usual in more recent times.

A few statistics on the subject may be given here with a view

[1] This chapter has already appeared more fully in vol. 87 of *Trans. Roy. Med.-Chi. Soc. of London.*

[2] *Sydenham Society's Transactions*, vol. ii. p. 850 *et seq.*

of showing the variability existing between the results obtained by treatment at different periods. It is interesting to recall the high percentage of so-called cures which have been recorded in the pre-bromide days; thus Hufeland[1] stated that a cure was effected in 5 per cent. of his cases. Russell Reynolds[2] noted that 10 per cent. of his cases were cured; Trousseau obtained 20 cures out of 150 cases, or 13 per cent.; and Herpin[3] was perhaps justified in his sanguine belief when he recorded 19 cures out of 38 cases, or 50 per cent. It would seem to be obvious from Voison's statement that Herpin's were really cases of permanent cure, for he observed many of these patients ten years after Herpin's death, and found them free from fits.[4] These are the results recorded by physicians who used remedies (such as oxide of zinc and nitrate of silver) which have long ago passed out of common practice.

The almost universal administration of the bromides, since their introduction by Sir Charles Locock in 1857 in the treatment of this disease, has not materially altered the variability in the results obtained. To mention a few instances:

Nothnagel	gives	from 4 to 5 per cent. of cures.
Laehr	„	6 per cent. of cures.
Aekermann	„	7·6 „ „
Dana[5]	„	from 5 to 10 per cent. of cures.
Wildermuth	„	8·5 per cent. of cures.
Habermaas[6]	„	10·3 „ „
Alt	„	12·5 „ „

Much of the discrepancy existing in statistics depends upon the definition of a "cure," the interpretation differing widely in nearly every writer's statements. Reynolds regarded only those cases as cured in which there had been perfect restoration to health during a period of at least four years after cessation of the seizures; but many authors fail to state what is their definition of cure, or recovery, from epilepsy, and in the majority of instances so-called "cures" are merely long remissions occurring spontaneously, or induced by suitable medicinal remedies.

[1] Hufeland, *Manuel de Méd.*, 1841. [2] R. Reynolds, *Epilepsy*, 1861.

[3] Herpin, *Du Pronostic et Traitement*, etc., 1852.

[4] Mentioned by Nothnagel, *Ziemssen's Encycl.*, vol. xiv. 1878.

[5] Dana, *Text-Book of Nervous Diseases*, New York, 1898.

[6] Habermaas, *Allg. Zeitsch. f. Psych.*, vol. lviii. p. 243, from which the other references are taken.

THE PROGNOSIS BASED UPON THE RESULTS OF TREATMENT.

In analysing the cases collected for the purpose of ascertaining the prognosis, I adopted certain guiding principles, and the following eliminations were made:

1. All cases which had not been under constant observation and treatment for a period of at least two years.

2. All cases which showed any co-existing complication, such as hemiplegia, albuminuria, or gross cerebral lesion.

3. All cases of pronounced idiocy or dementia.

By observing these restrictions, cases of idiopathic epilepsy were as far as possible selected; while any transitory amelioration, resulting from medicinal or other treatment, was checked by fixing the minimum period of observation at two years.

In the study of the cases the statistical method has been adopted, and the results have been recorded in percentages, the total number of cases used in the construction of each table being also given. Although many objections may be urged against such a method, it has been deemed to be the best available, while the results show a surprising uniformity when considered from various points of view.

The cases have been subdivided into three groups—arrests, improved, and confirmed—according as they have responded to treatment.

(*a*) The term "arrest" has been used at the outset advisedly in preference to "cure," owing to the uncertainty in defining the latter term. No case has been regarded as arrested, which had not been free from fits for a period of at least two years. (*b*) The cases classified as "improved," are those which have responded more or less satisfactorily to treatment. Under this heading are also included those cases in which a remission, sometimes for a number of years, has occurred, but in which a relapse has eventually taken place. (*c*) "Confirmed" cases are those which have shown a steady, though not necessarily a progressive, tendency to mental deterioration, without any material lessening either in the frequency, or the severity of the seizures.

As it is desirable to ascertain how far epilepsy is a disease which may be arrested, improved, or become confirmed, it is proposed to study the cases which have been collected, and the influence of treatment upon them, under various headings, so as

to define, as far as possible, the specific factors upon which a prognosis may be based.

The prognostic bearing and value of the following influences will therefore receive separate consideration.

1. The influence of a hereditary disposition.
2. The influence of age at the onset of the disease.
3. The duration of the disease.
4. The frequency of the seizures.
5. The character and time of the seizures.
6. The influence of sex.

1. The influence of a hereditary disposition.

For the purposes of the investigation, a family predisposition to epilepsy only was noted. The influence of the neuroses, such as chorea, migraine, alcoholism, and insanity, has been omitted from the statistics.

Table 31, *giving the percentage of hereditary and non-hereditary cases.*

	Arrests.	Improved.	Confirmed.	Total Cases.
	Per Cent.	Per Cent.	Per Cent.	
With hereditary history -	32·0	31·0	36·0	87
Without hereditary history -	33·6	47·2	19·2	125
				212

From this table it is seen that there is practically the same percentage of arrests in those with, as in those without, a hereditary history; the latter, moreover, showing a greater percentage of improved cases, and a decidedly smaller percentage of cases which eventually became confirmed.

The general prognostic conclusions, which may be drawn from these cases, are:

(*a*) That there is as great a chance of arrest of epileptic fits in those who have, as in those who have not, a known family history of epilepsy.

(*b*) In those who have a hereditary history the chances as to whether the fits become arrested, improved, or confirmed are in any given case about equal.

(*c*) That as regards general improvement, more is to be expected in those who have no hereditary disposition, while a considerably smaller percentage of confirmed epilepties is to be found amongst those who have no family predisposition to the disease.

2. The influence of age at the onset of the disease.

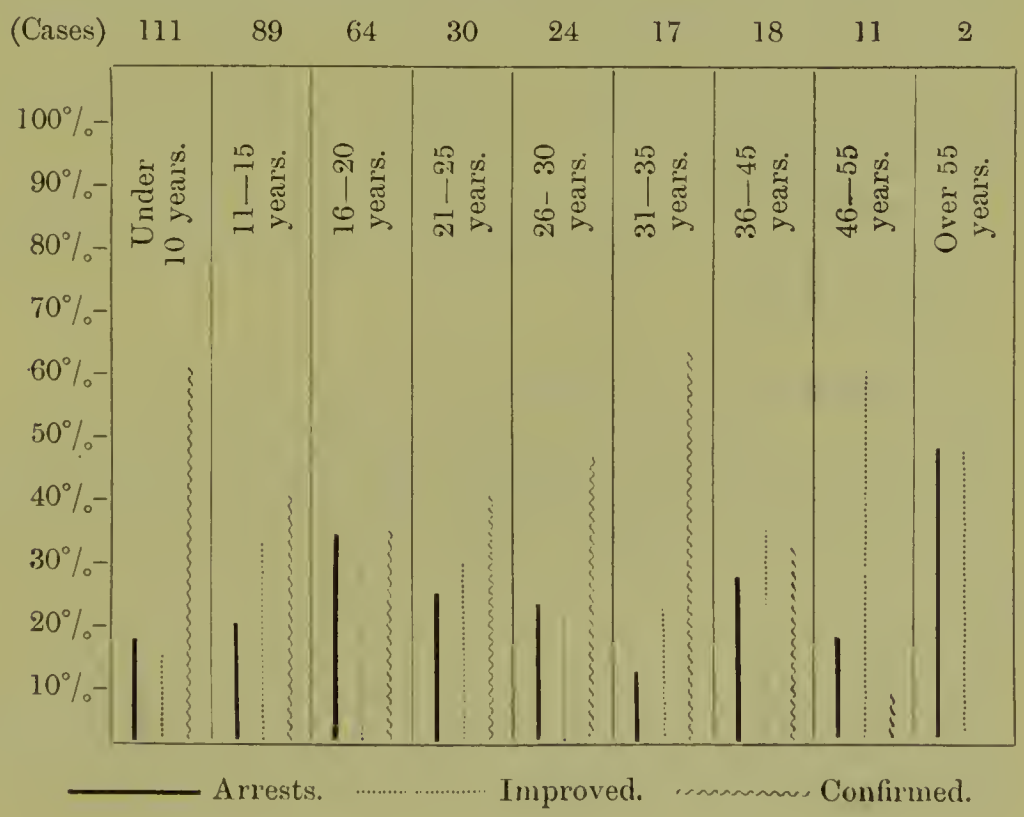
Table 32 shows the age-percentage at the onset of the fits, arranged in quinquennial periods, over ten years of age.

Age at Onset.	Arrests.	Improved.	Confirmed.	Total Cases.
	Per Cent.	Per Cent.	Per Cent.	
Under 10 years - -	19·8	18·0	62·0	111
11 to 15 „ - -	20·0	35·9	43·8	89
16 „ 20 „ - -	34·3	29·6	35·9	64
21 „ 25 „ - -	26·6	30·0	43·0	30
26 „ 30 „ - -	25·0	25·0	50·0	24
31 „ 35 „ - -	11·7	23·5	64·7	17
36 „ 45 „ - -	27·7	38·8	33·3	18
46 „ 55 „ - -	18·1	63·6	9·0	11
Over 55 „ - -	50·0	50·0	—	2
				366

The accompanying chart (Chart 12) is of value, as it shows that the age at onset of the disease is particularly important in considering the prognosis and facilitates the comprehension of the figures in the above table.

Chart 12.

Total number of cases, 366.



The main conclusions derived from a perusal of the above may be stated as follows:

(*a*) Epilepsy commencing under 10 years of age is least favourable as regards arrest or improvement, and most favourable for the production of confirmed cases.

(*b*) Those cases in which the onset of the disease is between 16 and 20 years of age show the greatest percentage of arrests and the lowest percentage of confirmed cases. From this quinquennial period onwards to that of 31 to 35 there is a steady diminution in the percentage of arrests, and a progressive increase in the percentage of confirmed cases.

The chief point of practical importance deduced from these figures, if put in general terms, is that epilepsy arising during puberty is not an intractable disorder, but that of adolescence is less amenable to treatment. These facts and figures confirm in a striking manner the opinion of Hippocrates, who wrote: "Epilepsy which commences about puberty is susceptible to cure, while that which comes on after 25 years of age as a rule only terminates with the patient."[1]

(*c*) From epilepsy arising during the quinquennial period, 31 to 35 years (which provides the least tractable form except perhaps that commencing under 10 years of age), there is a steady diminution in the number of cases which become confirmed, so much so that of those cases which arose during the decennial period, 46 to 55 years, only 9 per cent. became confirmed epileptics.

(*d*) Epilepsy arising over 55 years of age, to which the name of senile epilepsy has been applied by some writers, is essentially a tractable disorder.

3. The influence of the duration of the disease.

The term "duration" signifies the course of the disease from its commencement until the patient came under regular observation and treatment, from which time there dated either arrest of the fits, improvement, or a steady downward deterioration.

[1] *Aphorisms*, Section 5, No. vii. The aphorism is rendered in the *Sydenham Society's Transactions*, vol. ii. p. 738, as follows: "Those cases of epilepsy which come on before puberty may undergo a change, but those which come on after 25 years of age for the most part terminate in death."

Table 33, *giving the duration, percentage of the disease up to the commencement of treatment, and the general result of treatment, arranged in decennial periods.*

Duration.	Arrests.	Improved.	Confirmed.	Total Cases.
	Per Cent.	Per Cent.	Per Cent.	
10 years and under - -	23·5	32·3	44·1	272
11 to 20 years - - -	25·8	19·3	54·8	62
21 „ 30 „ - - -	12·5	20·8	66·6	24
31 „ 40 „ - - -	20·0	—	80·0	5
Over 40 „ - - -	50·0	—	50·0	2
				365

The table shows, by division into decennial periods, that there is as great a percentage of arrests when the disease has lasted from 10 to 20 years as from 1 to 10 years, but that under 10 years the percentage of improved cases is greater, and there is less tendency for the disease to become confirmed. But as ten years is a prolonged period, and as the majority of epileptics come under observation and treatment before so long a time has elapsed, it is important to ascertain the percentage frequency for periods short of ten years, and this has been done in the following table:

Table 34, *giving the percentage frequency in four unequal periods under* 10 *years of age.*

Duration.	Arrests.	Improved.	Confirmed.	Total Cases.
	Per Cent.	Per Cent.	Per Cent	
Under 1 year - - -	29·1	38·8	31·9	72
1 to 3 years - -	29·8	32·1	39·0	87
3 „ 5 „ - -	20·4	29·5	50·0	44
5 „ 10 „ - -	11·5	27·8	60·8	69
				272

Some general conclusions may be drawn from a perusal of these tables, as well as from a study of the subjoined chart (Chart 13), which gives in a graphic form the percentage results already detailed.

(*a*) Speaking in general terms, the earlier a case is brought under systematic treatment the more hopeful the prognosis and the greater the probability of improvement.

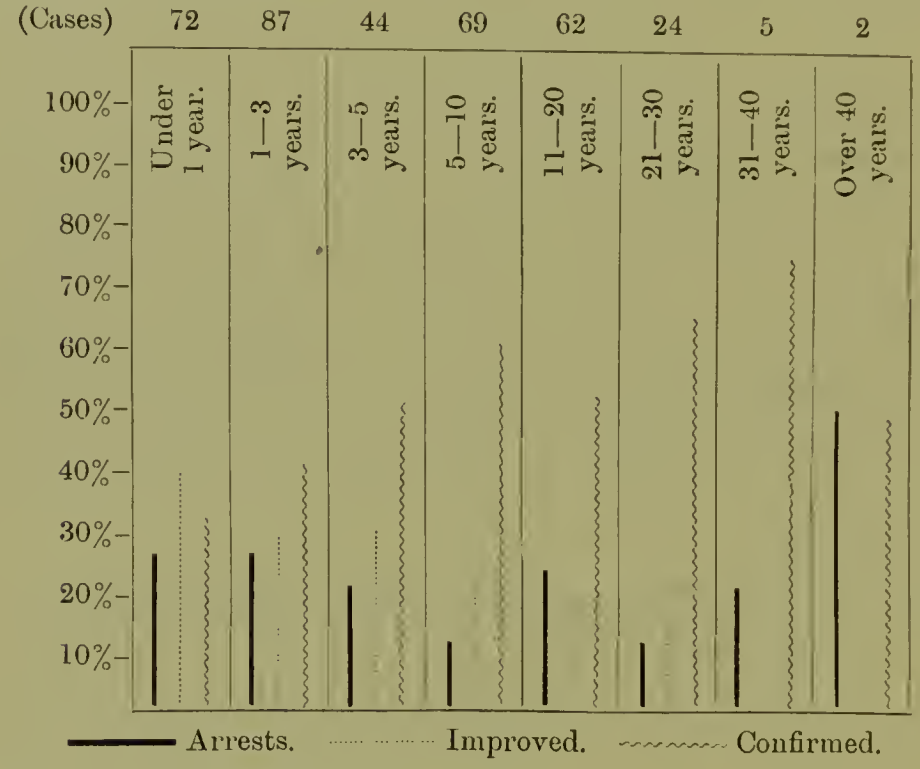
(*b*) There is a greater prospect of arrest or improvement during the first five, than during the second five years of the disease.

(*c*) Arrest of the fits, however, may take place in cases even after a duration of from 20 to 30 years. After 30 years arrest is possible, but the fewness of the cases hardly allows of any definite conclusions.

(*d*) There is a progressive tendency for epilepsy to become confirmed the longer the disease lasts without definite treatment.

CHART 13.

Total number of cases, 365.



4. The frequency of the seizures.

The relative frequency of the attacks has an important bearing upon the prognostic outlook, as will be readily seen from the following table:

Table 35, *giving the percentage frequency of the attacks.*

FREQUENCY.	ARRESTS.	IMPROVED.	CONFIRMED.	TOTAL CASES.
	Per Cent.	Per Cent.	Per Cent.	
Daily (1 or more) - -	—	42·5	57·5	40
Weekly (1 or 2) - -	12·5	23·9	63·5	96
Monthly (1 or 2) - -	22·9	31·2	45·8	96
Quarterly (1 or 2) - -	36·9	24·6	38·4	65
Yearly (1 or 2) - - -	42·1	47·3	10·5	19
				316

The general conclusion may be drawn that the longer the interval between the attacks the greater the prospect of arrest or improvement. Very infrequent attacks are eminently favourable. Attacks which occur every three or four months, or once or twice a year, are—within certain limits, and when considered in association with the points already mentioned in previous paragraphs—of more satisfactory prognostic importance than those which may be counted by the month, the week, or the day. The greatest percentage of confirmed cases, and the smallest percentage of arrested cases, are seen from the above table to occur in those epileptics who are subject to daily and weekly attacks; and the converse also holds good in that the smallest percentage of confirmed, and the highest of arrested cases, are found amongst those epileptics whose fits occur so infrequently as once or twice a year.

5. The character and time of the seizures.

The kind of attack to some extent modifies the prognosis. It is a matter of common knowledge that the major attacks are more readily influenced by drugs than the minor seizures. Owing to the incompleteness of the note in describing the exact character of the fits, it has been found impossible to construct a table of any real value, but so far as information has been supplied, it is clear that the greatest percentage of arrests is to be found in cases of the major type (49 per cent. out of a total of 96 cases); then follow the cases of combined major and minor fits (35 per cent. out of a total of 56 cases); and lastly, minor epilepsy occurring alone (26 per cent. out of a total of 15 cases).

So also with regard to the time-incidence of the seizures. Those occurring by day only—including in this the very common early morning seizure—give a greater percentage of arrests (51·9 per cent. out of a total of 52 cases) than those occurring only during sleep (34 per cent. out of a total of 35 cases). Combined day and night attacks give also an arrest percentage of 34 per cent. out of a total of 35 cases.

6. The influence of sex.

Sex plays little part in the prognosis of epilepsy. The following table will show the percentage of arrested, improved, and confirmed cases in this relation. From this it is seen

that rather more males show arrest of the seizures, but at the same time this sex gives a greater percentage of confirmed cases.

Table 36 shows the sex percentage of arrested, improved, and confirmed cases.

	Arrests.	Improved.	Confirmed.	Total Cases.
	Per Cent.	Per Cent.	Per Cent.	
Males - - - -	26	22	51	179
Females - - - -	20	34	44	187
				366

These results are in harmony with those of several previous observers (Reynolds, Gowers, and others), but are in opposition to the statement of Herpin.

II. THE CURE OF EPILEPSY.

Most neurologists are aware of cases in which a cure of epilepsy has occurred, and not a few instances may be gathered from amongst neurasthenics, who state that they suffered from fits in earlier years; and if the later histories of epilepties could be traced many more instances might no doubt be added. Although writers are generally agreed as to a cure of epilepsy, there is a less general consensus of opinion as to what is the definition of a cure—that is to say, after what period of arrest a "cure" may be said to have taken place. Before attempting to answer this question it is necessary to refer again to a few points to which attention has already been directed. With this object in view a table has been constructed dealing especially with the cases of arrest and of long remission, which are reproduced side by side for purposes of ready comparison.

The arrest column shows that the greater number of the cases were observed over periods of from two to nine years, during which no fits occurred; while of the remission cases, although four showed an arrest from four to five years, in five a relapse occurred up to eight years, after which time only one was found to relapse. I have, therefore, thought it *unsafe to regard as cured any case of epilepsy in which the seizures have been in abeyance for a period of less than nine years* after the disease has become satisfactorily established. This provision is made in order to eliminate all those cases of remission during childhood,

which are known to last for seven, eight, or nine years, and also those cases in which long intervals elapse between the first and second, or second and third, fits at the commencement of the disease. In order to obtain the percentage of cures in the present series, those cases only have been taken which were under observation for a period of at least nine years. They form a total of 147, of which 15 were arrested for nine or more years, *giving a percentage of* 10·2 *cures.*[1]

Table 37, *giving the cases of arrest and remission, with their duration, from amongst* 366 *cases of epilepsy studied for the purpose.*

11	cases of arrest,	1	of remission,	from	2	to	3	years' duration.
18	,, ,,	2	,,	from	3	to	4	,,
10	,, ,,	4	,,	from	4	to	5	,,
11	,, ,,	2	,,	from	5	to	6	,,
5	,, ,,	2	,,	from	6	to	7	,,
8	,, ,,	1	,,	from	7	to	8	,,
8	,, ,,	0	,,	from	8	to	9	,,
4	,, ,,	0	,,	from	9	to	10	,,
5	,, ,,	0	,,	from	10	to	11	,,
2	,, ,,	0	,,	of	11			,,
2	,, ,,	1	,,	of	15			,,
1	case ,,	0	,,	of	22			,,
1	,, ,,	0	,,	of	25			,,
86		13						

Although it may be laid down as a general rule that a cure of epilepsy has been established after an arrest of the fits for nine years, the fact must be borne in mind that a small percentage of cases do relapse after that period.

While discussing the cure of epilepsy, a point of practical importance, which should not be overlooked, is that those who have been cured of their seizures not infrequently show various mental peculiarities, such as impairment of memory, irritability of temper, and a tendency towards neurasthenic symptoms.

General conclusions upon prognosis.

A *neuropathic family history* will be found more frequently amongst those who have become confirmed epileptics, and who show the profounder degrees of mental impairment, but such history does not necessarily militate against the prospect of

[1] If arrest of fits for 8 years is taken as the definition of a "cure," the percentage of cures works out at 11·9.

arrest of the seizures, or improvement in the disease, or the retention of normal mental faculties in any given case. Direct parental heredity to epilepsy or insanity is associated with more pronounced stigmata of degeneration than collateral heredity.

The *age at onset* of the disease is of importance in prognosis. The most unsatisfactory cases are those in which the disease commences under ten years of age; they afford the smallest percentage of recoveries and the largest percentage of confirmed cases. They also present a high percentage with profound mental impairment, and a large proportion show structural stigmata of degeneration.

The most satisfactory cases are those in which the disease commences in the quinquennium, sixteen to twenty years. They present an almost equal percentage of arrested and confirmed cases, and there is as much chance of the mental condition being slightly, as profoundly, impaired.

If the disease commences between twenty-five and thirty-five years of age, a high percentage become confirmed epileptics; but from this decennium onwards there is a steady increase in the expectations of arrest and a diminution in the number which become confirmed.

The *duration of the malady* influences the prognosis to the extent that arrest or improvement is more likely during the first five than during the second five years. Similarly, mental impairment is less frequent when the fits have lasted less than five years, and mental integrity is less common when attacks have persisted for ten or more years. Epilepsy may be arrested even after a duration of twenty or thirty years, and mental health and mental impairment are observed when the disease has lasted, respectively, for over twenty years and under five years. The absence of stigmata does not necessarily imply an early or favourable termination, nor does the presence of stigmata militate against a satisfactory prognosis.

The *character-combination of the seizures* plays an important part in prognosis. The major attacks are the most tractable, then follows the type characterised by the combined major and minor seizures, and least favourable the minor seizures occurring alone. With the major type alone mental soundness is as frequent as mental impairment; the minor type is found with the lesser grades of mental deficiency, while the combined major and minor attacks show the profoundest degrees of

dementia. These also show the more frequent occurrence of stigmata.

The *frequency of the attacks* materially influences prognosis; the more frequent the seizures the greater the percentage of confirmed cases, and the greater the degree of mental impairment, and *vice versa*. Fits recurring in series are accompanied by a high grade of dementia.

Long remissions, induced either by successful treatment, or from spontaneous arrest of the fits, sometimes lasting for several years, are not uncommon. They are of favourable prognostic value, but are not synonymous with a cure of the disease.

A *cure of epilepsy* may be defined as arrest of the seizures for a period of eight or nine years, when it will be found that from 10 to 12 per cent. of cures may be expected.

CHAPTER XII.

THE TREATMENT OF EPILEPSY.

Prophylaxis—Treatment at onset of the disease—The bromides; their administration—Other remedies—Miscellaneous methods—Satisfactory cases—Organotherapy—Serotherapy—Treatment during the fit—Status epilepticus—Of the post-paroxysmal phenomena—Hygienic treatment—Education—Dietetic treatment—Institutional treatment—Surgical treatment—Epilepsy and life assurance.

TREATMENT OF EPILEPSY.

The treatment of so protean a malady as epilepsy requires to be considered from several points of view. In the older treatises, the management of the disease was directed to the subjection of the convulsive seizures by medicinal means; but within the last decade or two the epileptic has received more general attention, particularly in relation to treatment in special institutions.

The study of the natural history of epilepsy, rendered possible by the daily observation of epileptics over prolonged periods in such institutions, has brought into notice some features of the malady, which are held to be as important as the paroxysmal convulsions.

There is no single specific remedy, or system, in the treatment of idiopathic epilepsy. Satisfactory results are only to be obtained by a careful study of each patient and by the adoption of a combination of methods. These consist in the administration of suitable medicinal remedies and attention to the physical and mental condition, in the early stages, and the care and supervision of those, in whom the disease has become confirmed. The treatment should be commenced at the earliest possible time, after the onset of the convulsive seizures, and should be continued for long periods, extending

over at least two years, even in the most satisfactory cases. For this purpose treatment is best conducted in institutions, or under skilled supervision, by which means the mental, and bodily functions can be regulated and submitted to suitable forms of work, exercise, and dietary. In this way also general hygienic rules and practices, such as the regulation of work and rest, of amusements, of baths, and the like, may be most fitly carried out.

The treatment of epilepsy therefore merits consideration under the following headings:

1. The prophylaxis of epilepsy, or the recognition and treatment of the neuropathic child.

2. The management and treatment of young epileptics at the onset of the disease, embracing the medicinal and dietetic treatment of epilepsy and its several manifestations.

3. The hygiene of the epileptic.

4. The education of epileptic children.

5. The care of the confirmed epileptic—"Epileptic Colonies."

6. The surgical treatment.

1. The prophylaxis of epilepsy, or the recognition and treatment of the neuropathic child.

The prophylactic, or preventive, treatment of epilepsy can only be carried out with any certainty of success in childhood and youth. Children, who have inherited a neuro- or psycho-degenerative disposition, present certain mental and bodily characteristics, which should give the key-note to their management.

The symptoms are many and varied. In the convulsions ascribed to dentition, and in those which occur during the first two or three years of life as a result of pyrexia, intestinal worms, or other reflex conditions, are found danger signals of the utmost value, as indicating a tendency to convulsion, which may in later years develop into epilepsy, under the stress and strain of physical or mental over-exertion, of puberty, of child-bearing, or of trauma.

Allied to infantile convulsions, and equally important from the medical and hygienic standpoint, are such neuroses of childhood as night-terrors, head-banging and teeth-grinding, sleep-walking, nocturnal incontinence of urine, sleepless-

ness, outbursts of temper, choreiform movements, and the "habit-spasms"; and in addition to such definite objective phenomena, abnormal timidity, shyness, and self-consciousness may be seen.

The nervous symptoms of the neurotic child have their physical counterpart. Some of these children exhibit no defect of the bodily development, but many are thin, pale, and delicate. Errors of refraction are frequent, and are often to blame for apparent mental obtuseness and the recurrence of headaches.

Structural stigmata of degeneration are less likely to be extensively present in early childhood; but the symptoms above described are sufficient evidence of the inherited neuropathic tendency.

The management of the child, in whose family a tendency towards epilepsy and insanity exists, is based upon general hygienic and dietetic lines, and for its successful issue requires the co-operation of parent, teacher, and physician. Everything should be done to soothe and quiet, regulated periods of exercise and instruction alternating with periods of rest and repose. The avoidance of corporal punishment and chastisement by slaps and blows upon the head should be insisted upon.

Over-exertion and strain, whether in the class-room or playground, have to be avoided. The physician and the teacher rarely see eye to eye in these matters, so that it often becomes a matter of importance whether the neurotic child is not better cared for under private tuition.

Much may be done by the avoidance of all forms of diet of an exciting or stimulating character. Milk should form the staple dietary for many years, and tea and coffee should only be taken, if indeed at all, in small quantities. As these children are usually delicate and pale, and sometimes much below the average weight, cod-liver oil should be a regular article of diet during the winter months.

Drug treatment should only be resorted to when special symptoms arise requiring their aid. Such symptoms have been already described, and the physician has a remedy of great value in the bromide salts combined with simple food, fresh air, and cod-liver oil.

Long periods of rest and freedom from mental strain, with

holidays by the seaside, are the best aids in restoring and strengthening the nervous system of the hereditarily neuropathic child.

2. The treatment of epilepsy at the onset of the disease.

It has to be borne in mind that epilepsy is, in the majority of cases, a progressive degenerative malady, and that the object of treatment does not lie only in an attempt to combat the convulsions by sedative medicinal remedies, but to prevent by every possible means the tendency to mental deterioration, which is so important a clinical feature of the disease.

Although treatment in an institution is probably the most satisfactory method of managing those afflicted with this disease, it is not advisable to carry it out in every case; more especially in the early months or years of the disease, it is desirable to treat either at home or under the direction of a person skilled in the management of such cases.

There are three lines of treatment, alone or in combination, available at the commencement of the disease:

(*a*) Medicinal.

(*b*) Dietetic.

(*c*) A combination of the medicinal and dietetic with general hygienic treatment.

Medicinal treatment—*the bromides.* There is no single specific remedy in the treatment of epilepsy, although the alkaline salts of bromine come nearest to this definition.[1] But the influence of the bromides upon epileptic convulsions is variable and uncertain.

In the first place, bromide medication may arrest the seizures immediately, or within a short period of their administration, temporarily or permanently. In this division most of the curable types of epilepsy are found, cases characterised by an absence of mental impairment and with fits recurring only at long intervals—in fact, a mild type of the disease. If any given case is capable of arrest, a satisfactory response will be apparent within a comparatively short period of the commencement of the bromide treatment, as will be seen from a study of

[1] The bromides form the basis of most of the quack remedies used against epilepsy. An analysis of some of the more common is given in Appendix B.

the following table, which shows the influence of the bromide salts upon 86 cases of arrested epilepsy:

Table 38, *showing the result of treatment by bromides in* 86 *cases of arrested epilepsy.*

Arrest under 1 year's treatment	- - -	44 cases	or 51·7 per cent.
„ „ 2 years' „	- - -	9 „	or 10·5 „
„ „ 3 „ „	- - -	6 „	or 7·0 „
„ „ 4 „ „	- - -	4 „	or 4·7 „
„ „ 5 „ „	- - -	3 „	or 3·5 „
„ „ 6 „ „	- - -	2 „	or 2·3 „
„ over 6 „ „	(17-22 years) -	17 „	or 20·0 „
An uncertain case - - -	- - -	1	
	Total - -	86	

From this it is seen that of 86 cases, in which the fits were arrested for periods varying from two and a half to twenty-two years, rather more than 50 per cent. yielded to treatment within the first twelve months of regular bromide administration. Out of 366 cases of epilepsy, 86, or 23·5 per cent., belonged to this class.

Under this heading are no doubt included several cases in which the disease consisted merely of a few fits occurring within a short interval of each other over a brief period, a variety of the disease to which reference has already been made, when the clinical types were under discussion (p. 105).

Secondly, the bromides may induce a lessening in the severity and frequency of the seizures. This is the common temporary result of bromide treatment, and is what may be confidently expected in the majority of cases in the early stages of the disease. Sometimes this change is effected by the arrest of the major seizures, the minor continuing; or the bromides may change the time incidence of fits, from the waking to the sleeping hours, or *vice versâ*. In the series of 366 cases of epilepsy in which bromide medication was continued over long periods, 105, or 28·7 per cent., belonged to this class.

Thirdly, the bromides may exert no influence at all upon the disease, or may even augment the frequency, or severity, of the seizures. In the series already quoted, 175, or 47·8 per cent., remained unimproved and became confirmed epileptics.

These numerical facts may be given in the form of a table,

showing the general results of treatment by the bromide salts in 366 eases of epilepsy:

Table 39 showing the general results of prolonged bromide medication in 366 cases treated at the Queen Square Hospital.

Cases of arrest - - - -	86	or 23·5 per cent.
Cases showing improvement -	105	or 28·7 „
Confirmed cases - - - -	175	or 47·8 „
Totals - -	366	100 „

These figures are in general harmony with the observations of some other writers on the subjeet, notably Binswanger,[1] who refers to the result of bromide treatment in the Stephansfeld Institute for Epilepties in the following table, although the total number of eases on whieh the observations were made is not stated:

Arrest of seizures during treatment -	23·3 per cent.
Diminution in frequency to one-half -	40·0 „
No material change - - - -	36·6 „

Hughes Bennett[2] has given the following figures from observations of 300 epilepties treated by the bromide salts:

Arrested cases - - - - - -	12·1 per cent.
Lessening in frequency and severity -	83·3 „
No change - - - - - - -	4·6 „

Legrand du Saulle[3] found the following results from bromide treatment in 207 eases:

Arrest for from 6 months to 4 years -	37·6 per cent.
Some improvement - - - -	9·2 „
No improvement - - - - -	53·1 „

The general conelusion drawn from a study of these figures is, that under the influenee of the salts of bromine, a large number of eases of epilepsy are temporarily "eured," or materially improved. It is, however, within the experienee of most observers that no eomplete reeord can be obtained of the total number of eases which are relieved by these means; as more especially amongst the hospital eases, there is a tendency, onee the fits have been temporarily arrested, for the patient to eease attendanee. Some of these persons again attend

[1] Binswanger, *op. cit.*, p. 383.

[2] H. Bennett, *Edin. Med. Jour.*, 1881.

[3] Legrand du Saulle; quoted by Binswanger (*op. cit.*).

after many years, either for a relapse of the seizures, or for some independent malady.

It would be an error to say that bromides are useless in the treatment of epilepsy in view of the generally favourable results obtained by most observers.[1] If 50 per cent. of the cases derive benefit from the administration of these drugs, then surely all cases, especially of recent onset, should be given the benefit of the drug for a time. It is not the bromide salts, as a remedy, but the method of their administration, which is harmful, and to this attention will be directed immediately.

The *physiological action* of the bromide salts consists in lessening the irritability of the central nervous system, and in exerting a subduing effect upon reflex activity and cerebral function. The potash salts of bromine also induce a slowing of the pulse and the action of the heart. In medium doses (30-60 grains) the bromides produce muscular fatigue, a slowing of the mental processes, dulling of the sexual function, and of the skin sensibility. In large doses (150-225 grains) the speech becomes slurred, there is abolition of the palatal and pharyngeal reflexes, while frontal headache, and a limitation of the power of thought, soon ensue. Salivation, lowering of the body temperature and of the pulse rate, catarrh of the stomach and of the respiratory mucous membranes are general bodily symptoms resulting from continued use of large doses of the bromides.

It is therefore obvious that the prolonged and injudicious use of the bromides may give rise to the toxic symptoms, known as *Bromism.* This condition is characterised by a blunting of the intellectual faculties, impairment of the memory, and the production of a dull and apathetic state. The speech is slow, the tongue tremulous, and saliva may flow from the mouth. The gait is staggering, and the movements of the limbs feeble and infirm. The mucous membranes suffer, so that the palatal sensibility may be abolished, and nausea, flatulence, and diarrhoea supervene. The action of the heart is slow and feeble, the respiration shallow and imperfect, and the extremities blue and cold. An eruption of acne frequently covers the skin of the face and back.

[1] Few neurologists in this country will be found to support Spratling's contention that if recovery takes place under the use of the bromides, it is in spite of and not on account of the drug (*Med. Rec.* N.Y., 1905).

Dosage. Bromide treatment should be commenced at the earliest possible time after the onset of the fits, as there is a greater prospect of arrest or improvement during the first five, than during the second five years of the disease, although arrest of the seizures may occur after a duration of twenty years. The administration of the bromides should be continued for a period, the duration of which is to be determined by a study of each case separately, but should not be less than two years. The dose usually given is, I think, too large. If benefit does not follow a daily dose of from 30 to 60 grains of one, or a combination, of the bromide salts, some other remedy or method of treatment should be sought for and applied.

The large doses sometimes prescribed—from 75 to 150 grains daily—although no doubt suppressing the seizures for a time, induce other and more serious phenomena, viz., those of bromism already described. Moreover, the bromides have an accumulative action. Laudenheimer has shown that an epileptic taking 10 grammes (150 grains) of bromide salt daily for eight days, only excreted 35 grammes during that period. The blunting influence of the bromides upon the cortical motor cells has also been demonstrated by the fact, that a considerably stronger electrical current is required to excite the cortical motor areas of dogs, which have been dosed by bromide of potassium (Albertoni).

Methods of administration of the bromides. Most physicians have their own methods of prescribing the bromides in epilepsy. As already mentioned, large doses are neither necessary, nor effectual in their results; but both the amount of the dose and the frequency and time of administration should be gauged by a study of individual cases.

(*a*) If the seizures are only nocturnal, one dose of 30 grains of either the potassium or the sodium salt, taken in a small tumbler of water at bedtime, is usually sufficient.

If, however, the attack occurs in the early morning, or at or about the time of rising—a frequent time for the incidence of the epileptic fit—the full dose at bedtime may be followed by a half dose (grs. 15) when the patient wakes.

(*b*) Should the seizures occur during the day, the full dose may be taken after breakfast, and repeated at bedtime; or the full dose may be taken at bedtime and a half dose after breakfast. In this way the maximum of 60 grs., or

the medium dose of 45 grs. per diem, may be continued for a prolonged period. Nothing is to be gained by prescribing 15 grs. three times daily after the usual meals. Long experience in the use of the bromides has taught me that the best and most satisfactory time for their administration is at bedtime. The omission of the bromide for one day per week is often accompanied by satisfactory results.

(*c*) The three common salts of bromine (sodium, potassium, and ammonium) are the most usually administered. Each is of value, but those of potassium and sodium are most efficacious, and may be prescribed alone, or in association, a method of administration favoured by some physicians. If prescribed in this way, the dosage of the combined salts should not exceed 45 or 60 grains in the twenty-four hours. The bromides of strontium and lithium are less useful, but may be given in smaller doses, or in combination with one of the other salts.

A combination of strontium bromide, and bicarbonate of soda was recommended by Pugh as the most efficacious means of maintaining the blood alkalinity, but I have not found it more useful than sodium bromide alone.

(*d*) The bromides may be given alone dissolved in water, or in conjunction with arsenic, nux vomica, or gentian, so as to form a mixture. The taste may be partially obscured by aid of camphor, chloroform, or peppermint water; or the syrup of Virginian prune may be added as a pleasant medium for their administration.

(*e*) Combinations of the common bromide salts (potassium and sodium) with other remedies, which may have some direct influence either upon the nervous or circulatory systems, have been from time to time recommended, and are found useful in some cases. A combination of the bromides with *digitalis* has been found very satisfactory in cases with low arterial tension, irregular action of the heart, or failing compensation with valvular disease; *chloral hydrate* may be added with great advantage in cases of prolonged serial epilepsy or of the status epilepticus; the bromides and the *glycerophosphates*[1] form a valuable combination in weak or debilitated cases, more especially in young women with anaemia or neurasthenic symptoms.

[1] Dana (*Monthly Cycloped. of Pract. Med.*, 1905, p. 385) recommends a combination of sod. brom., grs. 60; sod. glycerophosph., grs. 20, in the twenty-four hours.

Bechterew[1] recommends the conjunction of the bromides with *adonis vernalis*, and sometimes with *codein*.

A combination of a bromide salt with *borax* has been of service, where the bromides or borax, separately, have been of little use.

(*f*) Within recent years many new preparations with bromine, in synthetical combination, have been introduced: such are *bromipin* (bromine and sesame oil); *bromaline* (bromine and formaldehyde derivatives); *bromocarpine* (bromine and pilocarpine).

I have not found these remedies in any way preferable to the common bromide salts; bromipin is difficult to dispense but may be given satisfactorily *per rectum*; and the other two presuppose the auto-intoxication theory as the cause of the seizures.

Duration of treatment. The question as to how long bromide, or any other form of medicinal treatment, should be maintained, is not one upon which any rigid statement can be made. Some authorities maintain that treatment should be continuous for a period of at least two years after the last seizure; but the experience gained at the Queen Square Hospital points to a much longer period as necessary. Epileptics attend there for many years after the arrest of the fits, as they find that stopping the bromide, even for a short time, conduces to a return of giddiness or of "sensations," reminding them of their previous attacks. If nine years' freedom from fits is to be the gauge of a "cure" of epilepsy, then withdrawing the bromide under a period of five years, in those in whom the fits are arrested, would be injudicious. On the other hand, many persons in whom the disease has become arrested after a year or two of bromide treatment, remain free from attacks without the aid of any medicinal remedy.

The withdrawal of medicinal treatment therefore, in those cases in which the fits are arrested, should be carried out tentatively and by degrees, a dose of bromide being taken every other, or every second, night for a time before being finally dispensed with.[2]

In cases of *confirmed epilepsy* with mental deterioration,

[1] Bechterew, *Neurol. Centralbl.*, 1894, p. 838.

[2] J. Voison (*l'Epilepsie*, 1897) finds the state of the pharyngeal reflex a good guide as to the elimination of the bromides.

the use of the bromides is of relatively little value. According to Féré the bromides are only of use when the dose is so large as to produce toxic effects, such as somnolence, mental inactivity, and loss of the palatal and pharyngeal reflexes. All that their continued administration in these cases is likely to produce may be a diminution in the number, and perhaps in the severity, of the seizures. With a view to test the efficiency or otherwise of a 30-grain dose of bromide salt administered every evening to a number of confirmed epilepties in an institution, the dose was stopped for a month, and the total number of fits with and without bromide was compared. The result was seen in an increase in the number of fits during the month without bromides from 278 to 402.

My experience of the treatment of confirmed epilepsy is that the administration of the bromides is of little real service, a slight reduction in the number of the fits being all that can be anticipated. On the other hand, any and every change of treatment will be of temporary benefit. Bringing the epileptic under the generally favourable hygienic surroundings of a hospital, will often conduce to material temporary improvement without the aid of any medicinal remedies. A reduction in the amount of the bromides taken during the twenty-four hours has not infrequently been followed by satisfactory results.

The most serious effect of sudden deprivation of the bromides in epilepties accustomed to their use, is a tendency to the development of the status epilepticus, in which the patient may die. Hence, even in confirmed cases, if it is deemed advisable to reduce the amount of the bromides given daily, the reduction should be carried out gradually.

Treatment by drugs other than the bromides. In the days before the introduction of the salts of bromine in the treatment of epilepsy, many and varied medicinal remedies[1] were used, sometimes with marked success, as may be seen from the satisfactory results obtained by Herpin, Reynolds, and others. On account of the not infrequent failure of the bromides to arrest, or even to ameliorate epileptic attacks, it will be found necessary to prescribe some other medicinal remedy, and a large number have been from time to time advocated and employed.

[1] The superstitious custom of wearing a silver finger-ring has been explained on the assumption that mercury (quick-silver) was formerly used as a remedy for epilepsy.

Perhaps the drug most frequently used as a substitute for, or as an adjuvant to, the bromides, more particularly in England, is *borax* (sodium biborate). Introduced by Gowers many years ago as an anti-spasmodic, it has met with considerable favour in cases where the bromides have been of little service. Unless in combination with a salt of bromine, I have not found it of particular use. It may be given in doses of from 10 to 30 grains thrice daily, but is apt to induce troublesome gastro-intestinal symptoms. If continued over long periods, it may lead to cutaneous eruptions of a psoriasis-like character.

Belladonna was the chief anti-epileptic remedy of the pre-bromide days, and is still used in some cases with marked benefit, when the bromides, or other remedies, have proved unsuccessful. It formed the chief remedy of Trousseau, Hufeland, Herpin, Reynolds, and others; and in the hands of the first-named was mainly used in those cases complicated with nocturnal incontinence of urine. A combination of bromide and belladonna may be found useful in cases of otherwise intractable combined-seizure types. It should be tried for a time in all cases in which the bromides have failed, for every now and again a case will be met with in which it produces remarkable amelioration of the seizures. It may be prescribed, either in liquid form, as the tincture in doses of 5 or 10 minims, or in the form of a pill made of the extract. or pulvis belladonnae. Recent investigations tend to show that the alkaloid *atropin* is without benefit in the treatment of epilepsy, although its employment, by subcutaneous injection at the seat of the local lesion, in cases of so-called reflex epilepsy is recommended, as it appears to have a dulling influence upon the irritability of the sensory and motor peripheral nerves.

The *zinc salts* (oxide, valerianate, and lactate) are old-established, and occasionally successful, remedies in the treatment of epilepsy, more especially in the hands of the French physicians.

Opium, as a remedy for epilepsy, dates from classical times. It is now only used in the *opium bromide method* recommended by Flechsig.[1] The principle of this method of treatment lies in the preparation of the nervous system for subsequent bromide

[1] Flechsig, *Neurol. Centralbl.*, 1893, p. 229.

medication by a preliminary administration of opium. One of the preparations of opium, preferably the extract, is given for a period of six weeks in increasing doses up to 15 grains per diem, when it is suddenly stopped and large doses of a bromide salt, from 90 to 120 grains, are substituted, this large dose being gradually diminished until about 30 grains are taken daily. The most favourable cases for its employment are young epilepties before or about puberty, and epilepsy in the early stages. Confirmed epileptics and those with pronounced mental deterioration are not materially benefited. Some authorities speak highly of this method, more especially Binswanger and his pupils, while others have not found its advantages outweigh its dangers. My own experience of the method has been limited and not satisfactory.

Strychnine has been recommended from time to time, and used with considerable success by some physicians.[1] In doses of gr. $\frac{1}{16}$ daily, it may be continued over considerable periods of time. Its *modus operandi* is probably merely as a nerve tonic, although it may have some influence in strengthening the tone of the vaso-motor centres. Strychnine finds its most useful application in the treatment of nocturnal epilepsy, especially where there is reason to suppose that the blood pressure is materially lowered.

Monobromate of camphor, eosinate of sodium, chloretone, and numerous other remedies have been tried, but without any special benefit.

On the basis of the auto-intoxication theory of the production of epileptic seizures, various *intestinal antiseptics* have been recommended and employed. The influence of a dose of calomel during the prodromal stage of single fits, or of serial epilepsy is usually beneficial, and should be given under any circumstances periodically to epileptics. Sulpho-carbolate of soda, salol, beta naphthol, salicylate of bismuth, and a host of other remedies of this character have been recommended and tried, alone and in combination with the bromides; but there is no evidence that the seizures have been arrested, or materially lessened by their use. As already shown, the auto-toxic theory of the causation of epileptic fits is by no means proven: and even admitting its occasional influence in some cases, it is only an auxiliary element in the evolution of the seizure.

[1] W. Tyrrell, *Strychnine in Epilepsy*, 1867.

The clinical features of the cases of epilepsy favourable and unfavourable for treatment.

Everyone who has had much practical experience of epilepsy knows how unsatisfactory the treatment of this disease is in a large number of cases, more especially in the later stages of the disease, when the bromides are of little or no avail. Moreover, most methods and systems of treatment are of some benefit for a time; but the progressive character of the disease and the tendency towards mental deterioration, in the course of years assert themselves, and the patient passes into the class of confirmed epileptics for whom little, or nothing can be done.

There is, on the other hand, quite a considerable percentage of cases, varying from 25 to 50 per cent., in which a favourable outcome may be expected, as a result of carefully considered and well-sustained treatment over a number of years. The following features, singly or in combination, may therefore be regarded as constituting a *favourable type* of the disease:

(*a*) Cases in which the disease commences between the ages of 16 and 20 years; for the statistics show that as many of these have the disease arrested as become confirmed epileptics.

(*b*) Cases in which the disease commences after the age of 45 years, in other words, the so-called "late epilepsy."

(*c*) Cases in which seizures are not more frequent than once or twice a year. This is an especially favourable feature, and its existence should prompt to continuous bromide medication for a number of years.

(*d*) The absence of any obvious mental impairment.

(*e*) The absence of numerous, or pronounced, structural stigmata of degeneration.

In contrast to the above, there are certain features which condemn the patient almost from the outset to incurability. Amongst the chief *unfavourable features* may be mentioned:

An early commencement of the disease, more particularly when under five years of age; great frequency of the seizures, especially when of the combined type; marked mental impairment and the "facies epileptica." Cases presenting the psychical equivalents or the post-paroxysmal psychoses, already described, are also included under this heading: while instances of serial epilepsy and of the status epilepticus are banded together as being most unsatisfactory.

Miscellaneous methods of treatment. It is not surprising that in a disease so resistant to treatment, and in which the exciting cause of individual convulsive seizures is so shrouded in mystery, that many diverse methods of treatment—other than the usual bromide and dietetic agencies—have been suggested, applied, and in turn thrown aside. It may, however, be useful to refer to some of them in this place, as a case, which has resisted one form of treatment, may react, for a time, satisfactorily to another. It should, on the other hand, not be forgotten, what is a well-recognised axiom in the management of epilepsy, that cases of this disease may respond favourably for a time to each and every change of treatment, medicinal or other, and even when active treatment is stopped. As the disease is characterised by spontaneous remissions in the frequency and severity of the seizures, a favourable result may occur, not on account of, but in spite of, therapeutic or other measures. With these facts in view, and with the knowledge that a cure of epilepsy implies arrest of the seizures for a number of years, a temporary benefit induced by any method of treatment ought not to be regarded too favourably until a sufficiently long period has elapsed.

Of all the recent systems, that which seemed likely to be of most use was the introduction of the organic extracts in the treatment of this disease; but further experience with these preparations has been on the whole disappointing.

Organotherapy. The administration of extract of the *thyroid* gland, or of *iodothyrin*, was at one time strongly advocated, more with a view to counteract the co-existent mental deterioration than as a subduer of convulsions. In a number of cases of confirmed epilepsy, in which preparations of the thyroid gland were given over considerable periods, no appreciable diminution was detected in the frequency of the seizures, and in only a limited number of cases, and for brief periods, was there any lessening of the co-existent dementia. My experience is that thyroid medication tends rather to increase the number of fits, and to produce at times irritability and want of control.

It has been already stated that preparations of the *thymus* gland act injuriously in epileptics by increasing the number of the seizures. *Cerebrin* has not been found to be of any value.

Serotherapy. The treatment of epilepsy by the injections of

blood serum, either from another epileptic, or by reinjection of the blood serum into the same epileptic, was introduced by Ceni,[1] who believed that a soluble epileptogenous poison existed in the blood serum in quantities varying with the severity of the disease. Ceni's view differed from that held by most supporters of the auto-toxic theory of convulsions, in that the amount of poison circulating in the blood did not change in the different stages of the disease. As the result of a number of observations, Ceni concluded that there exists in the blood of epilepties a soluble latent bio-chemical substance, an autocytotoxin, which has an influence over the elaboration of epileptogenic toxic agents, and produces its results after repeated injections. The records of these observations show that in some cases a beneficial, and in others an unfavourable, result followed the injection of the blood serum of epilepties into other epilepties.

The practical utility of this method in the treatment of epileptic fits, even in the hands of its originator, has not been sufficiently satisfactory as to render its application general; while later investigations, more particularly those of Sala and Rossi,[2] Gerharz,[3] and others, failed to confirm Ceni's results, or to establish any benefit at all from the injection of the blood serum of epilepties into others subject to epilepsy.

Treatment of the fits.

1. *To arrest the fit.* The first consideration may be given to the question as to whether it is possible to arrest an attack, once the warning has commenced. Many methods have been suggested for this purpose, some of which are of old standing and date from the time of Galen. The attacks in which abortive measures are likely to be successful are those commencing with a peripheral aura. The common method of encircling the wrist, for example, with a ligature or tape, and making traction upon it as soon as the aura is felt in the hand, is well known. As great force is sometimes required to arrest the attack, a strap is preferable to a tape or ligature.

[1] Ceni, *Riv. Sperimental. di Freniatria*, 1901, p. 344, and *Neurol. Centralbl.*, 1903, p. 388.

[2] Sala and Rossi, *Neurol. Centralbl.*, 1903, p. 852.

[3] Gerharz, *Neurol. Centralbl.*, 1904, p. 835.

Sometimes the patient alone is unable to produce sufficient compression, and requires the assistance of a second person. A circular blister was suggested by Buzzard, in order to induce a more permanent effect, sometimes with advantage.[1]

Forced extension or movement in the direction opposite to the warning sensation may be efficacious, when compression alone is unsatisfactory. According to Herpin,[2] the most effectual means of arresting such attacks is a combination of circular compression and forced movement in the opposite direction. Friction, or rubbing the extremity of the limb, where the sensation starts, has also been of use; and I have known a patient to bite the finger in which the aura commenced, sometimes with a successful issue.

Abortive means are less satisfactory in cases with a visceral aura. Strong pressure by the hands over the epigastrium is resorted to by some epileptics, while others prefer to drink cold water; swallowing a few drops of ether has also occasionally resulted in arresting an attack. Inhalations of ammonia have been used successfully. I recall a case in which a seton inserted over the epigastrium was employed with advantage in diminishing the frequency of the attacks. Other patients refer to a method of auto-suggestion, bringing to bear a strong determination to overcome the attack—a method which undoubtedly has been followed by success in some cases.

It is difficult to say how far the means employed have affected the arrest of the attack, or whether this has not, on the other hand, been spontaneous. We know that the incomplete, or minor, fit is merely the aura of the fully-developed seizure, so that an attack, thought to be arrested by mechanical means, as above described, may in reality only have been the aura without further development. Epileptics also tell us that they usually know, from the intensity of the warning, whether they are likely to be able to arrest its further progress: from which it may be inferred that the fully developed seizure has, as its warning, a more intense sensation than the incomplete attack.

All these methods may be of temporary use at the commencement of the disease; it is not long before they become ineffectual.

The inhalation of nitrite of amyl is a method of arrest,

[1] Buzzard, *Brain*, vol. iii. p. 554. [2] Herpin, *Accès Incomplet*, 1867, p. 29.

more especially valuable in fits with cephalic warning, and particularly in those with olfactory sensation (Gowers). Pierce Clark has insisted upon this remedy as a satisfactory means of aborting fits.

2. *During the seizure.* All that is usually required during the epileptic fit is to lay the patient on the floor, so as to obviate danger from falling. The collar or neckband should be removed and any constriction around the neck loosened. The discomfort from tongue biting may be prevented by inserting a cork or india-rubber ring between the teeth at the commencement of the clonic stage.

In fits occurring during sleep the danger of the patient rolling on to his face and suffocating is to be remembered. This can only be prevented by attention to the patient until the fit is over. Spratling found that 13 per cent. of epileptics with nocturnal seizures rolled on to their faces from the force of the convulsion.

The post-paroxysmal sleep should be encouraged, as it renders less severe the subsequent headache, which spontaneously disappears in the course of a few hours.

Placing the patient upon the left side (left lateral decubitus) at the onset of the seizure has been recently recommended as a satisfactory method of reducing the intensity of the fit. In the cases in which it was tried the fits were obviously shorter and less severe.[1]

Status epilepticus.

It has been shown that a gradually increasing number of seizures is the usual sign of the onset of the status epilepticus or of prolonged serial outbursts. With this warning the dose of bromide should be increased even up to double the quantity, and chloral (grs. 10-15) should be added, and the mixture repeated every four hours. Clark recommends the addition of a solution of morphia to the draught.

Should the fits be recurring with great frequency and severity, no remedy is of greater benefit than the inhalation of chloroform, given up to complete anaesthesia. On the other hand, in less severe types of status, or in serial epilepsy, a combination of a bromide salt (grs. 20) and chloral hydrate (grs. 10) may be repeated frequently (about every two or three hours) for a time with benefit, especially in the latter condition.

[1] M'Conachie, *Brit. Med. Jour.*, May, 1904.

The bromides given alone are of little avail; but within recent years their hypodermic administration in sterile solutions of not more than 10 per cent. (Clark) has been recommended; these may be repeated until 60 or 100 grains have been injected.

Injection of the bromides by means of *lumbar puncture*,[1] has also been advised, in sterile solutions of 30 grains to the ounce, 10 or 15 c.c. of the cerebro-spinal being withdrawn before 10 c.c. of the bromide solution are injected.

The hydrobromate of hyoscine has also been used occasionally with success (gr. 1/75 to 1/100 hypodermically).

In the post-convulsive stuporous stage of status epilepticus the treatment is that of acute dementia—strychnine, digitalis, and alcohol, if there is much cardiac debility. Abundance of nutritious food and careful nursing are essential features in the general management of this period.

Treatment of the post-paroxysmal phenomena.

1. *Acute dementia.* During the after-stage of exhaustion following upon ordinary seizures no special treatment is necessary, as the stage passes into that of sleep, from which the patient spontaneously recovers.

In the acute dementias following serial or status outbursts, on the other hand, great care and attention is required, the patient having to be nursed as one suffering from acute illness. It is during this stage that death may occur, a circumstance which is as frequently attributable to want of attention, as to the clinical condition. During the few days of stupor abundant and nourishing liquid diet, in the form of milk, eggs, and custards, should be frequently given. If the patient is unable to swallow, nourishment should be administered in the form of nutrient enemata. Hypodermic injections of strychnine (liq. strych. ms. 5 or strych. sulph. gr. $\frac{1}{30}$) may require to be frequently administered. If necessary, alcohol may be given in considerable doses, and the action of the heart steadied and maintained by digitalis or strophanthus. Later on, during the delusional stage, general attention and care is all that is usually needed, while later tonics may be prescribed with advantage.

2. *Acute mania.* As already described, this form of excitement, whether occurring as a post-paroxysmal phenomenon or as

[1] Morton, *Trans. Nat. Society*, U.S.A., vol. iii. p. 42.

a psychical equivalent, is characterised by the suddenness of its onset, the intensity and violence of its manifestations and the shortness of its duration, extending usually over a few hours. All that is therefore required lies in protecting the patient, and those attending him, from the effects of the violence and excitement. For this purpose resort may be had to the padded room. On the other hand, if a drug be considered advisable, I know of none more safe to administer, or more speedy, certain, and satisfactory in its action, than the hydrobromate of hyoscine in doses of $\frac{1}{75}$ to $\frac{1}{50}$ grain, injected hypodermically. One injection is usually sufficient to induce quiet and repose for a period of several hours.

3. *Automatism.* This requires no special line of treatment. If of genuinely epileptic nature, the attacks are usually short and resolve naturally; but if mainly of hysterical character, resort may be had to affusions of cold water, injections of apomorphine, or preferably the application of strong faradic currents through the wire brush.

Dietetic treatment.

In considering the question of diet in its relation to the treatment of epilepsy, two primary factors require consideration: (*a*) the probable nature of the condition leading to the characteristic periodic "explosions" of nervous energy, and (*b*) whether this condition may be in any way influenced by the quantity and quality of the food.

Many causes have been assigned as the immediate excitants of epileptic seizures, reference to which has already been made in the earlier chapters of this work; but the only certain factor is the unstable condition and proneness to "discharge" of the cortical nerve cells.

The researches upon the toxicity of the urine and the blood in epilepsy, although not completely harmonious, might be held to favour the toxic origin of some of the characteristic outbursts.

Owing, however, to the diverse views held upon the immediate exciting cause of epileptic seizures, and the variability of the results obtained from the practical application of different forms of diet, many authorities regard a rigid dietary as of little value in the treatment of epilepsy.

It is, however, not so much the quality of the food as its

composition, which should guide the physician in the selection of a suitable dietary. Attention should also be paid to the *quantity* of food ingested, for epileptics are notoriously big eaters, and, being habitually subject to constipation, are prone to overload the digestive tracts and organs.

In any attempt to formulate the principles underlying the dietetic management of epileptic fits, two views ought to be kept in mind: first, that nervous energy has its source chiefly in the albuminous and nitrogenised principles of foodstuffs (Hughlings Jackson). Secondly, that a paroxysmal toxaemia may be productive of some forms of convulsion, the toxic element being either introduced with the food, formed in the digestive tract, or arising from the metabolic changes which normally take place, more especially in the liver.

Guided by the first of these hypotheses, Merson[1] treated a number of epileptics upon farinaceous and nitrogenous diet alternately over a stated period, and noted the number of the seizures. The results of the treatment were not such as to justify the conclusion, that either kind of diet possessed any decided advantage over the other, in the treatment of epilepsy. As regards the number of fits in individual cases, there was a slight advantage in favour of the farinaceous regimen, the actual number of fits in the selected cases, over a period of four weeks, being slightly less upon a farinaceous than upon a nitrogenous diet. It is, however, a generally accepted doctrine that a diet rich in meat is more likely to induce an increase in the number of epileptic seizures than one consisting mainly of the farinaceous foodstuffs.

The experiences of Alt[2] upon the several forms of diet in epilepsy led him to the conclusion, that a diet without meat was the most satisfactory, but that neither a milk diet alone, nor a vegetable diet, was as beneficial as their combination.

"*Salt-starvation.*" It was an old speculation of Hughlings Jackson[3] that the part played by salts and minerals should be investigated in their relation to the building up of structure and the development of function. As the chlorides and the bromides were strikingly homologous in their chemical and physical properties, he suggested that the efficacy of the bromides

[1] Merson, *West Riding Asylum Reports*, vol. v., 1875.

[2] Alt, *Zeitschr. f. klin. Med.*, vol. liii.

[3] Hughlings Jackson, "Substitution Nutrition," *Ophthal. Hosp. Rep.*, 1866, p. 291.

might be due to their replacing, with greater energy, the more common chlorides.

The practical application of this suggestion, however, was not carried out in the treatment of epilepsy until Toulouse and Richet[1] recommended a diet in which the total quantity of sodium chloride *per diem* was limited to one or two grammes. It was thought that by diminishing the quantity of the chlorides the bromides might be administered in smaller doses, and the risks of bromism thereby lessened. The general result of the treatment has been to show, that in some cases "salt-starvation" has proved a useful adjuvant to bromide medication, while in others little benefit has resulted. Other observers have shown that the dietary is of especial value in cases requiring large doses of the bromides, and in those which show a ready tendency to bromide intoxication. On the other hand, it has been contended that the danger of bromide intoxication is greater, and that, if the diet is of any use at all, it merely acts as treatment for symptoms.

Various modifications of the original Toulouse-Richet method have been made from time to time by different investigators; and the dietary recommended by Balint[2] was the one adopted a short time ago in a series of observations which the author carried out.

The general result of this treatment in a series of cases of confirmed epilepsy was briefly as follows:[3] There were some cases in which the attacks were diminished during the continuance of the treatment, and others in which the improvement lasted after the diet had been stopped. These were cases in which the bromides were not well borne, or were even deleterious. There was reason to suppose that the minor seizures were not so satisfactorily controlled as the major, for in three cases the minor attacks were appreciably increased in frequency. The mental condition seemed to undergo some improvement. As a relief to dyspeptic symptoms it was found to be distinctly useful.

The above-mentioned results were obtained upon a diet which, in addition to the elimination of sodium chloride, contained no meat, fish, or fowl: so that on re-consideration

[1] Toulouse and Richet, *Acad. des Sciences*, 20. xi.

[2] Balint, *Neurolog. Centralblatt*, 1903, p. 347.

[3] Aldren Turner, *Rev. of Neurology*, 1904, p. 793.

of the subject I have been led to test the influence of a *purin-free* diet upon the incidence of epileptic seizures.

Influence of a purin-free diet.[1] The purin bodies, or substances constructed on a base, C_5N_4, are widely distributed amongst the common foods.[2]

Purin bodies exist in all forms of meat extracts, in both the white and the red meats commonly used as food, and in large quantities in certain glands, notably the thymus and the pancreas. They are also present to a less extent in some vegetables, and in tea, coffee, and cocoa.

A non-purin dietary may be selected from amongst the following common articles of food: milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, cauliflower, sugar, and fruit, both fresh and dried, and olive oil.[3]

The selected cases were examples of idiopathic epilepsy, which had been treated with little, or no benefit along conventional lines, by means of bromide salts: and they were divided for practical purposes into those of recent origin, and those of a confirmed type of the disease.

1. In the *recent cases of epilepsy* the substitution of a non-purin for an ordinary diet with a medium dose of bromide of sodium at bedtime, was followed by remarkable benefit in a number of instances. In one an increasing number of major seizures during five years, under the bromides and an ordinary diet, was followed by an arrest of the fits for a period of twenty months, which still continues. In another case, fits recurring about every two or three weeks gave place to a period of arrest for six months. In a third, numerous minor seizures, sometimes as many as twenty per diem, with an occasional major fit, were arrested for a period of two and a half years. In two other cases in which epileptic attacks had persisted for some months, notwithstanding bromide treatment, the substitution of a purin-free dietary brought about periods of arrest lasting ten months and seven months respectively.

2. A further series of observations were made upon a number of epilepties at the Chalfont Colony with a view to

[1] Aldren Turner, *Practitioner*, April, 1906.

[2] Walker Hall, *The Purin Bodies of Foodstuffs*, London, 1903.

[3] A list of common foodstuffs with their purin value will be found in Appendix C.

test the efficacy, or otherwise, of the purin-free diet in *confirmed epilepsy*, and to ascertain how far it was of service in the different clinical types of the disease. Amongst the cases under observation the following forms of seizure were observed: complete major attacks, incomplete minor seizures, psychomotor fits characterised by well-marked automatic movements, night and day fits, hysteroid seizures, and motor phenomena in the form of "jerks" and "jumps" and "falls," without subsequent convulsion. In general terms it may be stated that the majority of the cases felt better when on this form of diet, but that there was no obvious improvement in the quantity, or quality of the daily work which the patients had to perform. The only well-marked and definite change seemed to be a lessening in the severity and frequency of the convulsive seizures, more especially when these occurred in series. There was no modification in the character of the minor seizures, or of the psychomotor fits. If anything there was a tendency to an increase in the headaches from which epileptics with minor attacks are prone to suffer. These observations would therefore bear out what has been constantly noted in the treatment of epilepsy, viz., that in the confirmed disease, with frequently recurring seizures, little, if indeed any, benefit, is to be derived from treatment, whether medicinal or dietetic.

The *general conclusions* which may be derived from a study of the dietetic treatment are that certain dietary modifications are useful aids to medicinal remedies in the early stages, more especially when, owing to resistance to the ordinary bromide medication, the doses of these salts are increased to an excessive extent. When a purin-free diet is prescribed, or chloride of sodium is replaced by the bromides in the dietetic formulae, smaller doses of the bromides may be administered with advantage. The type of convulsion most favourably influenced is the major seizure, but little benefit is likely to be observed in the minor attacks, or the automatic phenomena commonly associated with them.

3. Hygienic treatment.

It is now necessary to indicate some general hygienic points in the treatment of epilepsy which should be carried out, not alone, but in conjunction with the prescribed medicinal and dietetic measures. In a disease so difficult to handle, and

so prone to mental deterioration, assistance from all points of view should be rendered; for no greater mistake can be made in the treatment of epilepsy than to rely solely on medicinal means, which so often fail to gain the desired end.

There are many cases of epilepsy, in the early stages of the disease, in which institutional treatment may either be impossible or inadvisable. To my mind these patients ought to be placed under the care of a well-trained and capable nurse-attendant. In this way only can the physician's instructions be satisfactorily carried out, in respect to suitable quantity and quality of food, the proper allotment of work and rest, and the carrying out of those physical and mental exercises consistent with the malady.

Dana[1] has emphasised the importance of violent physical exercise for a short period (20 to 30 minutes) three times a week, to be followed by a cool bath. Under any circumstances a certain amount of daily exercise in the open air is necessary: walking, running, tennis, cricket, and even football. It is scarcely necessary to point out that such exercises as bicycling, rowing, swimming, and riding should be avoided.

Epileptics suffer notoriously from lowered vitality and sluggish circulation in the extremities, for which warm baths, spinal douches, and massage are beneficial. The use of the hot bath in epilepsy is most necessary, not only for the ordinary ablutionary purposes, but to promote skin excretion, which is regarded by some as having a toxic character.

The choice of a profession, or an occupation, for an epileptic youth is often a matter of difficulty. An outdoor life is usually regarded as the most suitable, but whether it is likely to be beneficial entirely depends upon the physical vigour of the patient; hence farming in all its branches, or market-gardening, should only be recommended to those of robust constitution. All occupations fraught with danger to the person, such as handling machinery, are, upon the face of them, most unsuitable: but there are many semi-sedentary forms of work suitable for the frailer epileptic, such as drawing, modelling, and office work, bookkeeping and the like.

The epileptic need not differ from a normal healthy individual in his amusements. Only with regard to dancing should a word of caution be given, more particularly in the

[1] Dana, *op. cit.*

case of epileptic girls. I have frequently seen a fit ensue after a dance, either from the fatigue, or the late hours, which such recreation entails.

Alcohol in all its forms ought to be prohibited, and tobacco permitted to only a moderate extent. As explained under the dietary, tea and coffee are preferably avoided.

A question of importance may arise at the commencement of the disease should epilepsy develop during the school period. Ought the patient to continue his schooling, or not? If the disease has arisen during the period of mental strain preceding an examination, or if it be attended by mental dulness, complete abstinence from work for a time is enjoined by removing him temporarily from his studies. When the type of the disease has revealed itself, it is preferable that the education should be resumed under the care of a private tutor and medical supervision, or in a special school.

Education should be conducted along the lines laid down under the Education of Epileptic Children in all cases of epilepsy arising during childhood, more especially when accompanied by mental deficiency and backwardness.

The marriage of epileptics should be discouraged. A firm belief exists in the minds of many persons, that if an epileptic girl is married, her fits will be cured. Isolated instances in which this has occurred may be observed, but in the majority of cases, either no change at all ensues, or the fits are made worse.

4. The education of epileptic children.

It should be upheld as an axiom that epileptics require to be educated according to their individual mental capabilities. No greater mistake is committed in the management of young epileptics, than withholding from them the advantages of the mental and physical exercises entailed by educational methods under special supervision and direction. But it is of primary importance in this disease, as in the allied states of mental deficiency and backwardness, that the specially trained teacher and the physician should work together in jointly supervising the methods applied towards this end.

With the increasing attention which has been given within recent years to the care and management of epileptics, the

methods by which epileptic children should be educated have received special consideration. In England much is due to the interest shown by the public educational authorities in providing special schools and special medical supervision for those children suffering from epilepsy and defective mental development.

Shuttleworth,[1] who has made a careful study of the London School Board children under his care, found that the following subdivisions could be made out of 470 epileptic children whom he examined:

(*a*) 17 per cent. were cases of mild epilepsy, with infrequent fits and no obvious mental impairment, and were suitable to continue their study in the ordinary schools. These cases may be said to be of normal mental capacity, and are comparable to those classified under Group A in a previous chapter (p. 143).

(*b*) 27·5 per cent. showed some degree of mental impairment, and were preferably educated in special classes, as owing to their defective memory and the lesser degrees of mental deficiency, they were unsuited to compete with normal children. These correspond to Group B of the adult epileptics.

(*c*) 40 per cent. were capable of being educated along special lines, but owing to the frequency and severity of the seizures additional supervision was necessary. This group of cases, corresponding to Group C, already described in Chapter VI., were suitable for education in residential schools or colonies for epileptics.

(*d*) 15·5 per cent. were epileptic imbeciles or dements, and were incapable of education. These require supervision and care in the idiot asylums.

A classification along similar lines may be made in the cases occurring in private practice. Here, however, private tuition may be preferable to that obtained in special institutions.

The lines along which the education of young epileptics should be conducted therefore entirely depend upon the degree of mental enfeeblement in individual cases, but it should include physical as well as mental training. In the cases corresponding to Group B (slight mental impairment) some literary instruction may be advantageously given, accompanied by diagrammatic and pictorial representation of the subjects under consideration: but

[1] Shuttleworth, *Jour. of Mental Science*, 1904, p. 662.

in the lower grades (Group C) mechanical work along kindergarten lines is of most avail. (Shuttleworth.)

A form of educational treatment much in vogue in Sweden is the Sloyd system, a method which combines the education of the senses, the regulation and co-ordination of muscular movements, and the promotion both of the manual and mental activities through proper exercises (Shuttleworth). By aid of a series of gradational manual exercises the mind and the eye are trained to observation, the muscular sense is cultivated, and the power of concentration is notably stimulated. As described by one authority: "Sloyd is tool work so arranged and employed as to stimulate and promote vigorous, intelligent self-activity for a purpose, which the worker recognises as good" (Spratling).

The co-operation of all the senses is especially important in the education of these people; additions, subtractions, etc., ought to be demonstrated objectively. All forms of manual or industrial work may be advantageously prescribed—carpentry, mat-making, basket-making, sewing, gardening, kitchen and laundry work. Combined with work there should be a judicious admixture of games and play, and of outdoor and indoor recreations.

Another feature requiring medical co-operation is to be found in the benefits, which may accrue from the change of routine school work to others of a more pleasurable and less burdensome character, in certain classes of neurotic and epileptic children. The pedagogical view that because a child is able to work he is therefore fit to attend school, is not one which ought to be rigidly upheld on medical grounds.

5. Institutional treatment. Epileptic colonies.[1]

It is now generally recognised that a satisfactory method of treating epilepties both in the early and late stages of their disease is in special institutions.

Practical experience is against treating epileptics along with the hysterical and the neurasthenic in the wards of hospitals for nervous diseases, partly owing to the disturbance which an epileptic fit creates, and partly because of the occasional occurrence of severe post-paroxysmal mental phenomena or the status epilepticus, requiring special supervision and care at the hands of trained attendants.

[1] For further information upon colonies for epileptics the reader is referred to Appendix D.

Experience is also opposed to treating epileptics in the wards of asylums, or in infirmaries for the aged. In the early stages of the disease there is little or no mental deterioration, and in the later stages mental infirmity of the nature of dementia, rather than insanity, is the common type. Hence epileptic institutions should be provided, partly on the lines of a hospital and partly on the lines of an infirmary, with the necessary conveniences for treating epileptics as sick persons during certain complications, or accompaniments of their disease.

Epileptic institutions in this country are in the form of "colonies for epileptics," and have so far been used solely for the confirmed cases.

For the purposes of institutional treatment adult epileptics may be divided into:

1. Epilepties in the early stages of the disease, where there is little or no mental deterioration, and in whom the disease may be held in control by a combination of suitable medicinal, dietetic, and hygienic means (Class A, p. 143).

2. Confirmed epileptics, whose mental condition permits them to carry on all the common forms of work satisfactorily under direction (Classes B and C).

3. Epileptic dements, requiring special care and supervision, but who are not legally of the insane class (Class D).

An epileptic institution, therefore, is both a hospital for the treatment of patients in the early stages of the disease, and a "colony" for the care and management of those whose malady is confirmed.

The principles which guide the treatment of epilepties in a colony are:

1. Regular and congenial employment of a kind suited to the mental and physical condition of individual cases.

2. A suitably arranged and simple mode of life, with avoidance of excitement and abstinence from alcoholic liquors.

3. The judicious alternation of work and play, and the encouragement of the simpler forms of outdoor exercise.

4. The reduction of the usual sedative medicinal remedies to a minimal amount.

Outdoor forms of work are most suited to epileptics: sedentary employment, as in basket-making, bootmaking, and tailoring, has been found to be more conducive to the continuance of the seizures, than work in the garden or on

the farm. Housework and needlework for frail epileptic girls are satisfactory and beneficial, while the more robust among the women are capable of doing even heavy work in the laundry.

6. Surgical Treatment.

The favourable anticipations of the treatment of epilepsy, formed during the early years of Cerebral Surgery, have not been fulfilled. The cases in which a successful issue has followed an operation, either upon a peripheral portion of the body, or upon the cortex of the brain, are those in which the local exciting cause has played the chief rôle in the production of the disease. On the other hand, the cases in which surgical interference has led to disappointment, and they form the majority, are cases in which the hereditary element has had the larger share in causation. It has been already shown in an earlier chapter of this work, that over 50 per cent. of the cases of epilepsy owe their onset to hereditary causes, without local or exciting influences; and that in those having evidence of an exciting factor, the percentage with an hereditary history varied from 35·9 per cent., in the traumatic types, to 68·7 in the psychical. Hence a history of family epilepsy, or, of still greater value, objective evidence of neuropathic degenerative stigmata, is a feature upon which too great stress cannot be laid, when attempting to formulate an opinion as to the prognostic value of a contemplated operation. Moreover, the convulsive habit having been established in those of neuropathic tendencies by recurring convulsions, it is scarcely likely that the removal of a small portion of the cerebral cortex will entirely lead to their subjection.

The improvement, which the prolonged and judicious administration of the bromides is able to induce in many apparently confirmed cases of the disease, demands an extensive trial by medicinal means before resorting to surgical methods.

On the other hand, the occasional improvement brought about by operation, favours a resort to surgical interference in suitable traumatic cases in which the hereditary factor is not obvious, or, on careful inquiry, seems not to exist.

The importance of early operation in cases of the traumatic variety, in which fits ensue within a brief period of the

receipt of the injury, has been pointed out by Keen,[1] Cushing, and other surgeons, as likely to prevent the development of the convulsive habit and the establishment of the confirmed disease.

Cases of epilepsy, from the surgical aspect, resolve themselves into the following divisions:

1. Epilepsies associated with reflex (peripheral) irritation.
2. Jacksonian epilepsies, with or without local organic lesion.
3. Traumatic epilepsies.
4. Idiopathic epilepsy.

1. *Reflex epilepsies.* Under this heading are included those cases of epilepsy which appear to arise from local irritation of the peripheral organs, more especially the nose, eyes, ears, and genital organs, although no part or organ of the body is necessarily exempt.

I have shown on an earlier page that fits whose initial warning is referred to an organ of special sense, may be associated with a local peripheral lesion as a possible exciting or adventitious cause.

Foreign bodies, adenoid growths, polypi, etc., should be removed from the nose, and there are numerous cases on record of amelioration and arrest of seizures after such operations.[2]

Most observers refer to the great frequency of astigmatism amongst epileptics (Féré, Dodd, Gould). Work Dodd[3] has recorded the improvement, which was observed in a number of instances after suitable correction by glasses and the continuance of the medicinal treatment: though Gould and Bennett[4] are less sanguine as to the benefits likely to be derived from correction. In all cases errors of refraction ought to be corrected and suitable glasses prescribed and worn.

Coexistent disease of the ears and naso-pharynx require suitable treatment.

In some cases of epilepsy in boys, the removal of a tight prepuce has been followed by amelioration and arrest of the seizures.

Caries of the teeth requires the necessary attention, and I

[1] Keen, *Philadelphia Med. Rec.*, 1902.

[2] See St. Clair Thomson, *Practitioner*, May, 1905.

[3] Work Dodd, *Brain*, 1893, p. 534.

[4] Gould and Bennett, quoted by Spratling, *op. cit.*

have frequently observed improvement in the number and severity of epileptic fits after the adjustment of suitable artificial means of mastication.

In cases of epilepsy arising from traumatic lesions of the peripheral nerves, the source of irritation should be excised.

Dysmenorhœic conditions in women require the necessary attention. The fact that epileptic seizures frequently recur just before, or after, the menstrual period is no argument in favour of any local exciting cause being found in the pelvic organs. The old procedure of removal of the ovaries, or other portions of the female generative organs, was based on an entirely mistaken view of the nature and cause of epilepsy.

In all cases of epilepsy associated with the above-mentioned peripheral conditions, suitable medicinal treatment ought to be carried on, even after the correction or removal of the local disorders.

2. *Jacksonian epilepsies*, or fits commencing with localised convulsions, are characteristic of three types of morbid condition:

(*a*) Local organic disease of the cerebral cortex, its membranes or superjacent bone, such as tumours, cysts, syphilitic meningo-encephalitis, and paretic dementia. I do not propose to discuss the surgical treatment of these affections, as they lie outside the scope of this work.

(*b*) "Cerebral birth palsies," in which the seizures usually commence locally and are sometimes limited to the paralysed limb, or limbs, or involve one side of the body (hemi-epilepsy).

(*c*) A variety of idiopathic epilepsy in which the fits, usually at the commencement of the disease, begin locally.

The two last varieties may be considered together, as it is affirmed by some writers that many idiopathic epileptics, with local commencement of the fits, have suffered from an infantile cerebral palsy, of which no sign remains. The underlying lesion in these cases varies in character and degree from a local patch of chronic encephalitis with microgyria, to the most pronounced forms of cystic formation or porencephaly.

Removal of the focus of irritation, by excision of that

part of the cortex corresponding to the warning of the fit, has been recommended and practised; but the results have not been generally favourable, even in the early years of the disease and in young persons.

It seems hardly likely that the substitution of a scar, made by disease, for one made artificially, will lead to any pronounced amelioration of the seizures, especially as these cases are usually accompanied by considerable mental defect.

If an operation on the head is to be of any real value in cases of this nature, it should be performed at the commencement of the malady, Cushing[1] has, therefore, advised immediate trephining in cases of "birth palsies," and convulsions occurring during, or immediately after, parturition, and with the asphyxia of whooping-cough. He has shown that in a large number of infants, hemorrhage takes place readily from the small veins entering the longitudinal sinus. As infants bear the operation well, he is of opinion that early surgical interference will be the best preventive to the subsequent permanent paralysis and epilepsy which usually result.

3. *Traumatic epilepsies.* These epilepsies, as already shown, are primarily of two kinds, those with and those without organic lesion of the brain or its membranes (see p. 53). The majority of the latter type are cases of idiopathic epilepsy. Some of them, however, are cases in which, as the disease progresses, symptoms appear suggesting the presence of a local irritating lesion of the cortex cerebri. For the purposes of surgical consideration, epilepsy, ascribed to trauma of the head, may be subdivided into three varieties:

(*a*) Cases of idiopathic epilepsy, in which the trauma is merely an accidental and unessential circumstance in the causation of the disease. These cases do not require further consideration.

(*b*) Cases of convulsions arising from obvious traumatic lesion, such as occurs from fracture of the skull, gunshot wounds or other coarse damage to the brain. In these cases the question of operation has to be decided within a brief period of the infliction of the injury, and is of general surgical interest.

(*c*) Cases of epilepsy which at first appear to be of idiopathic nature, but later on show signs of a

[1] Cushing, *Amer. Journ. Med. Sciences*, Oct., 1905.

local irritative-paralytic lesion, indicated by the development of hemiplegic symptoms and the local commencement of convulsions.

The cases of the third class present the greatest difficulty. In the first place, the original fit may not ensue until some months, or even years, have elapsed after the injury. The early fits may be of a general convulsive character, with or without a local warning. Pure Jacksonian fits are rare; the occurrence of fits during sleep is not uncommon. The development of a post-convulsive, mono- or hemiparesis, eventually becoming permanent, is an important symptom.

Secondly, local signs over that part of the skull, which was the seat of the injury, such as a scar, a depression or prominence of the bone, or local tenderness on pressure are usually present.

In some cases of this type, removal of an ill-defined cyst, or scar from the cortex of the brain, has been succeeded by considerable improvement.

4. *Idiopathic epilepsy.* There were certain forms of operative procedure practised at one time, which have passed out of use, as they were based on erroneous conceptions of the nature of the disease. The most interesting was *excision of the cervical sympathetic ganglia.* The theory underlying this practice was that epilepsy arose from an anaemic state of the brain, a condition which was theoretically converted into one of permanent vaso-dilation, by paralysis of the vaso-constrictor nerves in these ganglia. A revival of this operation by Jonnesco[1] some years ago led to the collection by Winter[2] of a number of cases of epilepsy, in which it was performed. Of 213 cases, 91 were rejected as being of too recent a date to show satisfactory results. In the remaining 122 the following effects were noted:

4	cases arrested for 3 years, -	or	6·6	per cent.
17	„ „ „ 1 to 2 years,	„	13·9	„ „
23	„ improved, - -	„	18·9	„ „
67	„ showed no change, -	„	54·9	„ „
7	„ died, - - -	„	5·7	„ „

Judged by these figures this operation would appear to be as successful as many other forms of treatment in idiopathic epilepsy.

[1] Jonnesco, *Centralb. f. Chirurg.*, 1897, No. 2, and 1899, No. 6.

[2] Winter, *Arch. f. Klin. Chirurg.*, vol. 67, p. 816.

Ligature of the vertebral arteries has been entirely abandoned.

The insertion of a seton over the back of the neck, has some evidence in its favour; and in one case of epilepsy with epigastric aura, which has come under my observation, the statement was made that during the period of insertion of the seton, the fits were materially lessened.

Kocher recommends the operation of trephining with incision of the theca, under the belief that the disease is due to an increase of the intra-cranial pressure. There is, however, no clear evidence of the existence of such increased pressure in idiopathic epilepsy.

There are, however, some cases presenting fits, indistinguishable from those of true epilepsy, in which trephining may eventually become necessary. These are cases of gross intracranial disease, chiefly tumour, in which the earliest symptoms of the malady are generalised epileptic convulsions. Many of these cases are treated for years as epilepties; and only in the later stages evidence of progressive intracranial disease becomes obvious from the development of headache, optic neuritis, and a mono- or hemiplegic weakness.

There is another type of epileptic in whom opening the skull is indicated. I refer to a rare association of idiopathic epilepsy with cerebral tumour, usually gumma. One such case has come under my observation; the patient had his first epileptic fit when *aet.* 23 years; and these have continued to recur from time to time up to the present date. When *aet.* 27 he contracted syphilis. When he was 38 years old, he began to suffer from persistent headache, and about three months ago, when he first came under observation, double optic neuritis and a slight left-sided hemiparesis were detected. The later fits, observed when he was in the hospital, were Jacksonian in character, commencing at the left angle of the mouth and involving the left side of the face. A large gummatous tumour was removed from the right frontal lobe; and although he recovered from both the local and general symptoms referable to the tumour, the original epileptic seizures persist (now *aet.* 41).

Genuine idiopathic epilepsy does not lend itself to surgical operations upon the brain.

A brief **summary** may be added of the facts bearing upon

the reaction to surgical interference in the several types of epilepsy just described.

1. In all cases in which a reflex or peripheral irritation is observed to exist, its correction or removal should be carried out, as an essential correlative to medicinal treatment.
2. In the "cerebral birth palsies" with epilepsy and in the Jacksonian type of idiopathic epilepsy, operations upon the head fulfil no practical purpose. The subsequent development of epilepsy may be prevented in the former, if an operation is carried out at the onset, should the lesion be of the nature of a meningeal hemorrhage.
3. In the traumatic epilepsies, only those cases are likely to derive benefit from operation, in which a depressed fracture, a hemorrhagic cyst, or local encephalitis is present, and then only if it is carried out within a short time of the onset of the fits.
4. In general convulsions of an epileptic character a cerebral operation is of no avail, unless during the course of the disease general and localising symptoms ensue, pointing to the existence of a new growth in an operable locality.
5. In genuine idiopathic epilepsy, surgical interference is useless.

Epilepsy and Life Assurance.

Two points require consideration in this connection:

(*a*) The likelihood of epilepsy arising after insurance, in the offspring of an epileptic parent.

(*b*) The insurability of a person subject to fits.

(*a*) In the discussion of this question it may be noted that persons rarely present themselves for insurance under twenty-one years of age; and that as rather more than three-quarters of the cases of epilepsy develop before this age (780 out of 1000 cases, Table 6, p. 20), the prospect is largely in favour of a freedom from the malady, a freedom which increases as the years go on. According to Gowers[1] the chances are 1 to 50 against epilepsy in the hereditary cases developing after twenty, should the proposer have already

[1] Gowers, *Lancet*, Oct., 1904.

presented no evidence of the disease. A graduated addition to the premium would probably be sufficient protection in such cases.

(*b*) Ought an epileptic to be accepted for insurance? This is a matter which is viewed differently by insurance offices, some holding that they are uninsurable, while others carefully select their cases and protect themselves by a considerable additional charge.

By the favour of the Manager and Medical Adviser of the Scottish Equitable Life Assurance Society, I have been able to investigate the cases of epilepsy insured in that office for a number of years. The total number of accepted proposals, with a personal history of fits, during a period of fourteen years was twenty-three. It ought, however, to be stated that none of them was a confirmed epileptic, but only persons who had had at some previous period of life one or more epileptic seizures. The records were examined seven years later, when it was found that two persons had died, one from tumour of the brain, the other from uraemia. Of the remaining twenty-one cases, the eldest was 65, and the youngest 35 years of age. Agreeing for the present purpose with Muirhead, that the mean age at death in epilepsy is 48 years,[1] it was found that the average age of the twenty-one surviving cases was 47 years, or only one year short of the estimated mean age at death in epilepsy in insurance cases. It is therefore obvious that the acceptance of these cases was a justifiable procedure, more especially as the added years ranged from five to twenty.

The answer to the second question may therefore be given as follows: Some epileptics are insurable, more especially those whose fits are of infrequent occurrence, and those in whom the fits have been in abeyance for some time before insurance is proposed—a period of nine years' freedom being considered as a cure of the disease.

The dangers of sudden and accidental death in epilepsy have been already pointed out, and according to the statistics elsewhere quoted, are:

Sudden death as the result of a fit, - 5 per cent.
From accidents during a fit, - - 12 ,,

[1] Muirhead, *Scottish Widows' Fund, Report of Medical Officer*, 1902.

APPENDIX A.

Incidence and Mortality of Epilepsy among European Troops, Native Troops, and Prisoners in India for the Years 1899—1900—1901—1902—1903.

	1899.			1900.			1901.			1902.			1903.			Total.	Ratio.
	Strength.	Admissions.	Deaths.	Strength.	Admissions.	Deaths.	Strength.	Admissions.	Deaths.	Strength.	Admissions.	Deaths.	Strength.	Admissions.	Deaths.		
European Army—																	
Men - - -	67,697	86	1	60,553	81	1	60,838	125	1	60,540	139	1	69,613	95	2		
Women - -	3,093	5	0	2,908	2	0	2,729	5	0	2,555	9	0	2,891	11	0		
Children - -	5,500	4	1	5,376	4	0	5,069	1	0	4,709	10	3	4,677	2	0		
Native Army—																	
Men - - -	127,019	63	4	123,463	59	1	122,806	76	2	124,231	34	4	124,660	43	0		
Jail Population—																	
Men and Women	110,016	141	7	121,811	175	11	117,203	158	7	114,334	146	11	101,717	111	7		

I am indebted to Surgeon-General Branfoot, I.M.S., for these figures.

APPENDIX B.

Quack remedies for epilepsy.

The basis of all the patent remedies for epilepsy is bromide of potassium. The following are the formulae of the better known and most popular according to the analyses in the *British Medical Journal*, vol. ii., 1904, p. 1586:

Ozerine.

Pot. bromid. - - - - -	120 grains.
Am. carb. - - - - -	16 grains.
Burnt sugar.	
Chloroform water to one ounce.	

Dose—Four teaspoonfuls daily; amount of bromide taken per day is 60 grains.

Taylor's "Celebrated anti-epileptic mixture."

Tinct. iodi. - - - - -	¾ minim.
Pot. bromid. - - - - -	13 grains.
Am. bromid. - - - - -	4 grains.
Water to one ounce.	

Dose—One teaspoonful thrice daily; amount of bromide taken per diem is only about 6 grains.

Osborne's mixture.

Pot. bromid. - - - - -	166 grains.
Syrup - - - - - -	1 drachm.
Burnt sugar.	
Peppermint water to one ounce.	

Dose—One large teaspoonful twice daily; amount of bromide taken daily is a little over 40 grains.

Trench's remedy.

(1) Liquid form.

Pot. bromid. - - - - -	70 grains.
Am. Bromid. - - - - -	10 grains.
Sugar - - - - - -	72 grains.
Fuchsin.	
Water to one ounce.	

Dose—Three teaspoonfuls daily; amount of bromide taken daily is 30 grains.

(2) "Concentrated" form.

Pot. bromid. - - - - -	61 parts.
Moist brown sugar - - -	39 parts.

APPENDIX C.

The following is a list of the common foodstuffs, according to their purin value, for which I am indebted to Dr. Walker Hall:

Purin-free foods.—Milk, eggs, cheese, butter, sugar, white bread, rice, tapioca, cabbage, cauliflower, lettuce, macaroni, strawberries, olive oil.

Purin-poor foods.—Potatoes, onions, oatmeal porridge, French beans, turnips, carrots, parsnips, rhubarb, sea-kale, chickory, spinach, dates, figs, the pulses, asparagus, codfish.

Purin-rich foods.—Salmon, halibut, plaice, beef, pork, mutton, chicken, veal, liver, and sweetbread.

Beverages. *Purin-free.*—Port, sherry, claret.

Purin-poor.—Lager beer, pale ale, porter, tea, coffee, cocoa. China tea is the least rich in purin elements.

APPENDIX D.

Epileptic colonies.

A colony for epileptics should consist of:

1. Residential buildings of the villa type, constructed to hold from 18 to 24 or 30 inmates. Each building should be complete in itself in so far as domestic and sanitary matters are concerned. A competent head nurse should be in charge of each house. The houses for the male patients should be entirely distinct from those for the females, and preferably some distance apart. As the colony grows in magnitude additional villas may from time to time be added. The arrangement of the villas, whether in the form of a street or other plan, must of necessity depend upon various considerations, such as the value and extent of the land, the nature and fall of the ground, etc., so as to permit of effective drainage, water supply, etc.

2. A farm, market-garden, farm buildings, dairy, bailiff's house, etc.

3. Workshops, such as carpenter's, boot-maker's, tailor's, blacksmith's, etc. A laundry is essential, and affords employment for the female patients.

4. The schools should be either attached to the residences of the children, or in very close proximity to them.

5. A hospital is a necessary element of an epileptic colony. Many able-bodied epilepties become sick persons for short periods after severe attacks, and require, in some cases careful nursing and attention and in others restraint.

6. Recreation rooms and play ground are as necessary as workshops.

7. An infirmary for the old and demented would appear to become a necessity in a colony as time goes on. Colonies in England have not been sufficiently long established for this necessity to have arisen, but in the older colonies on the Continent it has been found preferable to transfer the aged and infirm to such a building rather than to an asylum or infirmary.

8. The administrative department, consisting of the residences of the superintendent and matron and the general offices and dispensary.

The financing of an epileptic colony. There are several ways by which money may be raised to support and maintain an epileptic institution.

1. *By payments from the educational authorities.* As the education of epileptic children is as much a matter for the educational authorities as that of normal individuals, the funds for this purpose may be legitimately provided by the School Boards. In the majority of cases it is advisable that epileptic children should be educated apart from the non-epileptic, and it is advocated that the schooling of epileptic children should be carried out in colonies for epilepties. A recent Act of Parliament—the Elementary Education (Defective and Epileptic Children) Act, 1899—authorises the School Board authorities to contribute, under certain conditions, towards the funds of voluntary institutions, such as the existing colonies, in order to permit the education of epileptic children being carried out.

2. *By payments from public authorities.* Owing to the detention in workhouse infirmaries of numbers of epilepties, many of whom are sane, able-bodied, and capable of work under direction, the Poor Law Guardians have the power to pay for and maintain such persons in epileptic institutions, where their services may be usefully employed and their disease treated

upon scientific lines. It is also in the power of Boards of Guardians to assist in maintaining epilepties in the existing colonies.

3. *By payments from the patients.* This is an important item, and a necessary one in the early stages of an unendowed colony. In some instances three grades of "colonists" are admitted. The first, a contributing class, pay sufficient to cover their own expenses as well as to assist those of the third class, who are most likely unable to meet the full outlay required for their keep. The second, or intermediate class, merely cover their expenses by their payments. In course of time the industrial, or farming, portions ought, under judicious care and management, to cover their expenses and afford a balance of profit; but in a colony in full working order, with the necessary superintendent, medical officers, heads of departments, instructors, nurses, and attendants, it is scarcely likely that the whole can be maintained without financial assistance from outside.

Epileptic colonies at present existent in the United Kingdom.

1. The Maghull Institute for Epilepties, near Liverpool, was founded in 1888. It is a colony whose chief object lies in employing adult epilepties, both males and females. The present number of inmates is about 204. The method of housing the inmates employed in this institution differs from that of other colonies in that there are two large houses, one containing men and the other women, instead of the usual villa residences.

2. The Meath Home, near Godalming, Surrey, was founded in 1892. Only female epileptics are admitted to this home, which is capable of giving residence to about 80 patients.

3. The National Society for the Employment of Epileptics has a colony situated at Chalfont St. Peter, Buckinghamshire, twenty-one miles to the west of London. Its present epileptic population numbers 200. It consists of eight villa residences containing from 18 to 24 inmates, situated on a farm of 270 acres: there are also all the necessary attributes of a large and increasing colony, such as workshops, laundry, dairy, market-garden, recreation room, and playing fields. The colony was founded in 1894.

4. The David Lewis Epileptic Colony, Alderley Edge,

Manchester, has 94 resident epileptics, but accommodation for 178.

5. There is a colony at Epsom, Surrey, in connection with the Asylums of the London County Council, which contains 325 patients. This colony differs from the preceding, in that the patients are certified as insane under the Lunacy Act.

6. A small epileptic colony has been recently founded at Bridge of Weir, Scotland.

INDEX

Abdominal reflexes in, 88.
Abortive treatment, 239.
Abscess, 49.
Absinthe-epilepsy, 7, 63.
Accidents in, 160.
Acute epilepsy (*see* Status epilepticus), 5, 101.
Adonis vernalis, 233.
After-stage of fits, 86.
Age at onset of, 18.
 and heredity, 30.
 and prognosis, 215.
 mean age at onset, 22.
 mental condition and, 147.
Albuminuria in, 190.
Alcohol and epilepsy, 3, 63.
 alcoholic epilepsy, 63.
 delirium tremens, 63.
Alcohol in treatment of, 242, 249.
Amblyopia in, 91.
Ambulatory automatism, 131.
Amusements for epileptics, 249.
Anæmia of brain, 7.
Anæsthesia in, 71, 91.
Angeiomata, 179.
Antiseptics (intestinal) in, 236.
Astigmatism, 36.
Atropin in, 235.
Auditory aura, 81.
Aura, 68.
 epigastric, 75.
 fear, 76.
 head sensations, 77.
 of special senses, 79.
 peripheral, 73.
 psychical, 77.
 visceral, 75.
Aura sensations, 68, 139.
Aural haematoma, 161.
Aural vertigo, 209.
Auto-intoxication in, 8, 199.
 symptoms of, 200.
 theories of, 200.
 types of epilepsy and, 201.
Automatism, 84, 126.
 ambulatory, 131.
 as equivalent states, 130.
 in alcoholics, 132.
 in hysteria, 133.
 medico-legal aspects, 126.
 post-convulsive, 84, 126.
 treatment of, 243.
Auto-suggestion in, 240.

Baths in treatment of, 248.
Belladonna in, 235.
Blindness as aura in, 80.
Blood in, 192.
 coagulability of, 193.
 constitution of, 192.
 corpuscles, 192.
 leucocytosis of, 192.
 parasites in, 196.
 plates, 193.
 reaction of, 194.
 toxicity of, 195.
 tumours, 179.
 vessels (cerebral), 178.
Borax in, 235.
Brain, tumours in, 60.
 treatment of, 258.
Bromaline, 233.
Bromides, 227.
 combinations of, 232.
 dosage of, 231.
 duration of, 233.

Bromides, general results of, 229.
in confirmed epilepsy, 233.
methods of administration of, 231.
physiological action of, 230.
with other remedies, 232.
Bromipin, 233.
Bromism, 230.
Bromoearpin, 233.
Brown-Séquard's fits, 7.
Burns in, 161.

Carbamate of ammonia, 196.
Carotid arteries (ligature of), 6.
Catatonic stupor, 139.
Cephalic sensations, 77, 139.
Cerebellum in, 180.
atrophy of, 180.
intravascular clotting in, 180.
Cerebral stigmata, 35.
Cerebro-spinal fluid, 196.
lymphocytosis of, 198.
Cervical sympathetic, 7, 257.
Character of epilepties, 119.
Chemical pathology of, 188.
Chloral hydrate in, 232.
Cholin in, 196.
Chorea and, 157.
Circle of Willis, 185.
Classification (general) of, 65.
according to mental state, 143.
Clonic convulsion, 85.
Clotting in cerebral vessels, 8, 182.
Coagulability of blood in, 193, 200.
Codeia, 233.
Colonies for epilepties, 251, App. D.
Combined type of, 97.
Convulsive factor in, 4.
Corneal reflex, 88.
Cornu Ammonis, 173, 175.
sclerosis of, 173.
Course of epilepsy, 110.
Cry in epilepsy, 83, 85.
Curability of epilepsy, 220.
Curable cases of epilepsy, 228.
Cure of epilepsy, 220.
definition of, 220.
Cyto-toxins in, 239.

Daily rhythm, 106.
Deafness, 92.
as aura, 81.
Death in epilepsy, 116.
causes of, 116.
mean age at, 117.
Decubitus (left lateral), 241.
Definition of, 1.
Delirium tremens and, 63.
Delusional states (transitory), 125, 138.
Dementia in, 10, 122, 142, 152.
as integral feature, 152.
pathology of, 10.
permanent, 142.
temporary acute, 122.
treatment of acute, 242.
Dental anomalies, 33.
Dental caries and, 254.
Dentition, 33.
Determining causes of, 41.
Diagnosis of, 66, 202.
Diet in epilepsy, 243.
Differential diagnosis, 204, 208.
Digitalis in, 232.
Diphtheria, 50.
Dislocation of shoulder, 161.
Dreamy state in epilepsy, 79, 135.
as aura, 79, 136.
as equivalent, 135.
as paroxysmal psychosis, 122, 135.
Drug habit and, 3.
Duration of, and mental conditions, 146.
prognosis, 216.

Ear disease and epilepsy, 62, 254.
Ears of epilepties as stigmata, 34.
Ecchymoses in, 161.
Education of epilepties, 249.
Emphysema of skin, 161.
Enteric fever, 49.
Epilepsia cursiva, 78.
Epilepsy and insanity, 26.
Epileptic insanity, 121.
Epileptiform convulsions, 207.
Epochal epilepsies, 21.
Equivalents, 4, 126.
Etiology of, 12, 41.
Errors of refraction, 37, 254.

Exercises in, 248.
Exophthalmic goitre and, 159.
Eye disease and epilepsy, 37, 62, 254.

Facial asymmetry, 32.
Facies epileptica, 150, 162.
Fall in epilepsy, 83, 160, 203.
Fractures in, 161.
Frequency of seizures in, 111.
and mental condition, 150.
and prognosis, 218.
Frequency of epilepsy, 15.
according to different writers, 16.
in the Registrar General's Reports, 16.
in the sexes, 15.
of insanity, 17.

Genital organs, 62, 254.
Glycero-phosphates in, 232.
Gout and, 3.
Grand mal intellectuel, 133.
Graves' disease and, 159.
Gumma cerebri and, 159, 258.
Gunshot wounds of head, 54.
Gustatory aura, 79.

Hard palate (deformities of), 33.
Headache, 86, 140, 156.
as aura, 77.
as substitution symptom, 157.
post paroxysmal, 86.
(paroxysmal) and epilepsy, 156.
Heart disease and, 164.
Hemorrhages in, 161.
Heredity, 2, 23.
and mental condition, 145.
and stigmata of degeneration, 37.
collateral heredity, 28.
frequency of hereditary factors. 2, 25, 26.
in hospital and private cases, 27.
percentage frequency of, 26, 30.
similar and dissimilar, 24.
statistics of, 25, 26.
Hereditary factors in, 2.
alcoholism, 25, 26.
epilepsy, 2, 25, 26.
insanity, 3, 25, 26.
other nervous disorders, 27, 29.
Hernia in, 161.
Herpes Zoster, 49.
Hydrobromate of Hyoscine in, 242, 243.
Hygienic treatment of, 247.
Hyoscine, hydrobromate of, 242.
Hyper-leucocytosis, 9, 193.
Hypnosis in, 207.
Hysteria and epilepsy, 3, 204.
Hysterical seizures, 205.
features of fit, 205.
sequelae of fit, 206.
Hystero-epilepsy, 5, 204.

Impulsions of epileptics, 126, 134.
exhibition, 126, 134.
gluttony, 135.
homicide, 135.
pyromania, 135.
sexual perversions, 126, 135.
suicide, 135.
theft, 135.
Infantile convulsions and, 29, 48.
Infantile eclampsia, 48.
Infantile hemiplegia and, 59, 255.
Infective disorders as cause of, 49.
Influenza, 49.
Ingestion of food and, 62.
Inheritance of, 2.
Insanity and epilepsy, 3, 26.
frequency of, 16, 18.
Institutions for epileptics, 251.
Intestinal worms, 62.
Intravascular coagulation, 8, 182.
Invulnerability of, 194.

Jacksonian epilepsy, 207.
diagnosis of, 207.
treatment of, 255.

Knee-jerks in, 87.
Kussmaul-Tenner fits, 6.

Labyrinthine vertigo and epilepsy, 208.
diagnosis of, 209.

Lactation, 46.
Laughter (paroxysmal), 141.
Leucocytosis in, 9, 192.
Life insurance and, 259.
Localised convulsions, 207.
Locomotor ataxy and, 159.
Loss of consciousness in, 66, 202.
Lymphocytosis in, 197.

Major epilepsy, 94.
 major fit, 69.
 mental state in, 95.
Malaria, 50.
Mania (acute), 124, 133.
 post-convulsive, 124.
 as equivalent state, 133.
 treatment of acute, 242.
Marriage of epileptics, 249.
Masked epilepsy, 127.
Massage in treatment of, 248.
Masturbation, 62.
Mean age at onset of, 22.
Measles, 49.
Medulla oblongata, 180.
Membranes of brain in, 170.
Memory, loss of, 119.
Meningitis, 50.
Menstruation, 43.
Mental states in epilepsy, 118.
 inter-paroxysmal, 142.
 paroxysmal, 120.
 temperament, 119.
Migraine and, 156.
Minor epilepsy, 96, 209.
 minor fit, 68.
 mental state in, 97.
 diagnosis of, 209.
Myoclonus epilepsy, 158.

Narcolepsy, 141.
Nasal disease and epilepsy, 61, 254.
Nerve cells in, 167.
 degeneration of, 167.
 in imbeciles, 169.
 persistence of, 169.
 vacuolation of, 168.
Neurococcus, 196.
Neuroglia, 10, 171.
Neurogliosis, 171.
Neuropathic associations of, 155.
Neuropathic child, the, 225.
Nitrite of amyl in, 240.
Nocturnal epilepsy, 47, 106.
Nucleo-proteid clotting, 89.
Nystagmus, 90.

Obsessions in epilepsy, 37, 121, 136.
Occupations for epileptics, 248.
Olfactory aura, 79.
Onset of epilepsy (age at), 18.
Onset of (methods of), 109.
Opium-bromide method, 235
Optic thalamus in, 178.
Organic epilepsy, 60.
Organo-therapy in, 238.

Paralysis in, 88.
 agitans, 159.
 exhaustion, 88.
 infantile, 59.
 motor, 88.
 nature of, 90.
 sensory, 91.
Paroxysmal psychoses, 120.
 post-paroxysmal, 122.
 pre-paroxysmal, 121.
Pathological anatomy, 165.
Pertussis, 49.
Perversions in, 126, 135.
Petit mal intellectuel, 134.
Physical condition in, 162.
Plantar reflex in, 87.
Pneumonia, 49.
Porencephaly, 59.
Pregnancy, 44.
Prevalence of, 12.
 in Australia, 14.
 in Brazil, 14.
 in France, 13.
 in Germany, 13.
 in India, 14.
 in Italy, 13.
 in Negroes, 14.
 in Russia, 13.
 in Switzerland, 13.
 in United Kingdom, 12.
 in United States, 13.
Prodromata, 70.
 explanation of, 72
 motor, 71.

Prodromata, psychical, 71.
sensory, 71.
somatic, 72.
vasomotor, 72.
Professions for epileptics, 249.
Prognosis of, 211,
and age at onset, 215.
and character of fit, 219.
and duration, 216.
and frequency of fits, 218.
and heredity, 214.
and sex, 219.
Prophylaxis of epilepsy, 225.
Pseudo-angina pectoris, 75, 140.
Psuedo-appendicitis, 75, 140.
Psychasthenia, 136.
Psychical causes of, 50.
Psychical epilepsy, 83, 130.
psychomotor fits, 84, 130.
Psychical epileptic equivalents, 126, 130.
definition of, 129.
Psychical factor in, 5.
Psycholepsy, 137.
Psychoses of epilepsy, 120.
post-paroxysmal, 122.
pre-paroxysmal, 121.
Puerperium, 45.
Puerperal eclampsia, 46.
Pulmonary tubercle and, 164.
Pulse in, 87.
Pupil in epilepsy, 88, 164.
Pupil in hysteria, 205.
Purin-free diet in, 246.
Purin-free foodstuffs, App. C.

Reaction of blood and, 194.
Reflex causes of, 61.
Reflex epilepsy, 61, 254.
Reflexes in epilepsy, 87.
Remissions, 112.
Reproductive functions in, 43.
lactation, 46.
menstruation, 43.
pregnancy, 44.
puerperium, 45.
quickening, 44.
Reproductive organs and treatment of, 255.
Rheumatic fever, 49.

Salt-starvation in, 244.
results of, 245.
Scalp wounds, 160.
Scarlet fever, 49.
Sclerosis of brain in, 171.
Senile epilepsy, 108.
Septic throat, 49.
Sequelae of fits, 81, 206.
Serial epilepsy, 98.
Sero-therapy in, 238.
Seton, use of in, 240, 258.
Sex and, 15.
and age periods, 17.
deaths from epilepsy in each, 16.
frequency in each, 16, 17.
and mental condition, 144.
and prognosis, 219.
Sleep, 47.
Sloyd system in, 251.
Sneezing (paroxysmal), 141.
Somnambulism, 132.
Spinal cord in, 181.
tract degenerations in, 181.
Stammering, 37.
Status epilepticus, 5, 101, 123.
death from, 105.
lumbar puncture in, 242.
prognosis of, 104.
relation to epilepsy, 5.
relation to serial, 101.
treatment of, 241.
Status hystericus, 104.
Stigmata of degeneration in, 3, 12, 30.
anatomical, 32.
and age at onset of, 38.
and clinical course, 3, 37.
and degenerative psychoses, 135.
and hereditary predisposition, 37.
frequency of, 35.
and mental states, 151.
pathological, 36, 37.
psychical, 37.
Stomach diseases, 62.
Strabismus, 90.
Strontium bromide, 232.
Strychnine in, 236.
Stupor, 123, 139.
as equivalent of, 139.
post-convulsive, 123.

Substitution phenomena in, 141.
Surgical treatment in, 253.
Sweat in, 198.
Syphilis and, 3, 55.
Syphilitic epilepsy, 57, 60.
heredity in, 58.
congenital, 3, 55.
secondary, 56.
syphilitic organic lesions and, 57.

Tangential fibres, 36, 167.
Teeth, disease of, and, 34, 254.
Teeth in epilepsy, 33, 62.
Temperament in epilepsy, 119.
Temperature in, 86.
Termination of epilepsy, 115.
Thrombosis in, 8.
Thymus gland in, 198.
Thyroid gland in, 238.
Tics and epilepsy, 37, 158.
Tobacco and epilepsy, 63.
Tongue biting, 160.
Tonic spasm, 85.
Toxaemia in, 200.
Toxaemic causes, 61.
Trauma in, 51.
Traumatic epilepsy, 52.
coarse lesions in, 54.
heredity in, 53.
treatment of, 256.
Treatment of, 224.
Tremor in epilepsy, 163.
Tuberculosis and, 3.
Tubercle of lungs, 164.
Typhoid fever, 49.
Types of epilepsy, 93.
combined type, 97.
major type, 94.
and mental condition, 149.
minor type, 96.
serial epilepsy, 98.
status epilepticus, 101.
with a few fits only, 105.
nocturnal and diurnal types, 106.
senile epilepsy, 108.

Uncinate fits, 79.
Uric acid, 189, 196.
Urine of epileptics, 188.
reaction of and fits, 189.
post-paroxysmal albumen, 190.
toxicity of, 190.

Vaccination, 49.
Vaso-constriction, 7.
Vaso-motor spasm, 7.
Visual aura, 80.
Vertebral arteries (ligature of), 258.

Weight, 92, 163.
Whooping-cough, 49.
Worms (intestinal) and, 62.

Zinc salts in, 235.

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